WESTERN EQUINE ENCEPHALITIS IN MANITOBA

MANITOBA

DECEMBER 1982

WESTERN EQUINE ENCEPHALITIS IN MANITOBA

ERRATA SHEET

After printing, several errors were detected which might lead to misinterpretation; these are noted below.

1. The proper citation for the report is as follows: "Sekla, L. (Ed.), 1982. Western Encephalitis in Manitoba. Manitoba Health Services Commission, 296 p.".
2. Page 11; delete Surgeoner, G.O., 1982.
3. Page 14; paragraph 4, number 1: replace period by a comma and add partially at the end of the line.
4. Page 22; paragraph 3, last line: "1982" instead of "1983".
5. Page 51; paragraph 3, line 1: change "indicating" to "indicates".
6. Page 64; Results, paragraph 1, line 6: delete "and" between week and increasing and insert a comma instead.
7. Page 87; Flock Locations, line 11: delete "before".
8. Page 88; Results, line 4: insert "4th" after August.
9. Page 88; Results, line 7: insert "of" between cases and WEE.
10. Page 99; Results, paragraph 2, line 6: insert "the remaining" between "in" and "sites".
11. Page 100; paragraph 2: start paragraph by "In 1981,".
12. Page 104; paragraph 2, line 7: insert "21" between 1981 and cerebrospinal.
13. Page 104; paragraph 3, line 2: change "3775" to "2775".
14. Page 122; last word on page: delete "in".
15. Page 122; may be missing in some copies. Write to the Editor for a copy of this page, if needed.
16. Page 123; paragraph 2, line 8 and line 11: "Vira A" should be corrected to "Ara A".
17. Page 195; equation 5: The denominator of the equation should be pi times the dynamic viscosity "squared".
18. Page 253; Recommendations 1, line 6: between "a" and "aerial" insert "properly designed monitoring program, firstly, to confirm that".
19. Page 256; paragraph 2, line 2: "presents" instead of "prevents".
20. Page 293 and page 294 are reversed.

MANITOBA

MINISTER OF HEALTH

WINNIPEG, MANITOBA
R3C 0V8

FOREWORD

Manitoba has experienced some recorded epidemics of Western Equine Encephalitis in 1941, 1975, 1977 and most recently in 1981.

In each of these years the staff of the Department of Health and co-operating agencies have done an in-depth evaluation of its activities during the epidemic, studying the factors which led up to it and the action taken. Each outbreak has led to improvements in technique, methods of monitoring and action. These have been published so that others may learn from our experience. Progress throughout history has been made by each generation "Standing on the Shoulders" of their forebears.

In 1975 I had the honour of establishing the Manitoba Arbovirus Surveillance Committee. In a public enquiry in 1982 the Clean Environment Commission stated that the decision making process of that committee "could not be improved upon", which is high praise, indeed. Albeit, my staff advises me that they have improved the program, as a result of the evaluation. In such ways progress is made.

It is my pleasure to endorse the publication of this monograph so that we, ourselves and others may benefit by our Manitoba experience.

T.R. Edwards

T. R. Edwards, C.A.
Deputy Minister

L.L. Desjardins

Laurent L. Desjardins
Minister

Note from the Editor

L. Sekla, M.B.B.Ch., D.T.M.&H., D.M., Ph.D.

Cadham Provincial Laboratory
750 William Avenue
Winnipeg, Manitoba
R3C 3Y1

Following the 1981 Western Equine Encephalitis (WEE) outbreak in Manitoba, the Provincial Government sponsored 2 reviews. The first one was a WEE Workshop held in February 1982; it included the presentation of formal, technical papers by members of the Manitoba Arbovirus Surveillance Committee (MASC), as well as a critical review of Manitoba's program by invited expert panelists and discussions in small groups, each specializing in one of the following 4 disciplines: i.e., Virology, Epidemiology, Vector Ecology, Control. The second review was a public hearing on "Mosquito Control Programs in Manitoba" conducted by Manitoba's Clean Environment Commission of Manitoba. It included submissions by experts as well as by lay persons.

Members of MASC felt that it would be beneficial to consolidate the data presented on both occasions and to preserve them in a book form similar and complementary to the WEE monograph published in the 1976 supplement of the Canadian Journal of Public Health. Funding for this publication was obtained from the Department of Health and authorization to publish excerpts from the Clean Environment Commission report obtained from the Minister responsible for Environmental Management. Our thanks go to both Ministers of Health and Environmental Management and to Dr. J. C. Wilt, for their help in this endeavour.

The task of obtaining the technical papers from the various authors was a laborious one; it was further complicated by the limitations in funding which forced MASC to select one of its members as an Editor. I am not sure I wish to thank MASC for selecting me. Though I tried to provide some homogeneity in the format and style of the papers, individual author's wishes were respected; this is particularly apparent in the nomenclature and abbreviations used for Western Equine Encephalitis and _Culex_ _tarsalis_, as well as in the legends for tables and figures. As much as possible, duplication of information presented in the Canadian Journal of Public Health was avoided.

I acknowledge with gratitude the tremendous help provided by the Editorial Board consisting of Dr. R. Ellis and Dr. R. Brust. Very special thanks are due to Miss Marie Myndzak who typed many of the manuscripts, some barely legible.

As well, I wish to thank Mr. Guenter Bormann of Manitoba Health Service Commission for his invaluable help in getting this book in print.

Finally, I wish to thank all past and present members of MASC for their various contributions. In 1981, the membership of the Manitoba Arbovirus Surveillance Committee was as follows:

Mr. J. Bates, Information Services, Room 29, Legislative Building,
Winnipeg, Manitoba. R3C 0V8

Dr. R. Brust, Department of Entomology, University of Manitoba,
Winnipeg, Manitoba. R3T 2N2

Dr. J. A. Eadie, Provincial Epidemiologist, 831 Portage Avenue,
Winnipeg, Manitoba. R3G 0N6

Dr. R. Ellis, City Entomologist, 2799 Roblin Boulevard, Winnipeg,
Manitoba. R3R 0B8

Mr. H. Fraser, Atomspheric Environment Service, 1000 - 266 Graham Avenue,
Winnipeg, Manitoba. R3C 3V4

Dr. W. G. French, Executive Director, Medical Public Health, 831 Portage
Avenue, Winnipeg, Manitoba. R3G 0N6

Dr. D. Gemmill, Medical Health Officer, City of Winnipeg Health
Department, 280 William Avenue, Winnipeg, Manitoba. R3B 0R1

Dr. G. Johnson, Deputy Minister of Health, Room 301, Legislative Building,
Winnipeg, Manitoba. R3C 0V8

Dr. J. Neufeld (Chairman), Veterinary Pathology, University of Manitoba,
545 University Crescent, Winnipeg, Manitoba. R3T 2N6

Mr. K. Plews, Environmental Control Programs, Building 2 - 139 Tuxedo
Boulevard, Winnipeg, Manitoba. R3N 0H6

Dr. L. Sekla, Assistant Director, Cadham Provincial Laboratory, 750 William
Avenue, Winnipeg, Manitoba. R3C 3Y1

Mr. D. L. Smith, Provincial Entomologist, 910 - 401 York Avenue,
Winnipeg, Manitoba. R3C 0P8

Please direct all correspondence regarding this publication to the Editor.

Table of Contents

Page

Foreword. Minister and Deputy Minister of Health, Manitoba 1

Notes from the Editor 2

Table of Contents 4

CHAPTER I Introduction 8

. Brief Historical Review of Western Equine Encephalitis - R. Brust 9 - 11

. Manitoba Arbovirus Surveillance Committee: Historical Review, Terms of Reference and Structure - L. Sekla 12

. Manitoba Arboviral Surveillance Committee - Description of the Surveillance Program - L. Sekla 13

. Health Emergency - The Decision Making Process - J. Eadie 14 - 17

. The Emergency Public Information Program - N. Donogh 18 - 19

CHAPTER II Mosquito Data 20

. Population Dynamics of *Culex tarsalis* Coquillet in Manitoba - R. Brust 21 - 30

. Summer Weather in Southern Manitoba, 1981 - H. Frazer 31 - 35

. Forecasts of *Culex tarsalis* Populations in Winnipeg, Manitoba - R. Raddatz 36 - 49

. Arbovirus Isolations from Mosquitoes in Manitoba: Value in Decision Making - L. Sekla and W. Stackiw 50 - 60

CHAPTER III Equine Data 61

. Western Equine Encephalitis in Manitoba - Equine Cases 1975 - 1981 - J. Neufeld and G. Nayar 62 - 79

. Investigation of Western Equine Encephalitis Activities in North Western Ontario 1981 - G. Surgeonner, W. Ralley, M. Mahdy 80 - 84

CHAPTER IV Sentinel Chicken Flocks Data 85

. Sentinel Flock Monitoring Procedures for Western Equine Encephalitis in Manitoba. 1976 - 1981 - F. Wong and J. Neufeld 86 - 97

. Use of Sentinel Birds in Evaluating Effectiveness of Aerial Adulticiding in Manitoba, 1977 and 1981 - J. Neufeld and F. Wong 98 - 101

CHAPTER V Virology 102

. Arbovirus Isolations and Serology in Manitoba - L. Sekla and W. Stackiw 103 - 116

CHAPTER VI Clinical and Epidemiological Data 117

. Clinical Study of Western Equine Encephalitis in 1981 - B. Friesen and J. Eadie 118 - 128

. Neuropsychological Sequelae After Western Equine Encephalitis Virus Infection - G. Hawryluk, G. Hammond, and B. Friesen 129 - 141

. Epidemiological Study of Western Equine Encephalitis in 1981 - J. Eadie and B. Friesen 142 - 155

CHAPTER VII Vector Control 156

. Insecticides Registered for Biting Fly Control in Canada - J. Hollebone 157 - 169

. Alternatives to Chemical Control of Mosquitoes in Manitoba - M. Chance 170 - 177

. Mosquito Abatement Programs in Manitoba - K. Plews 178 - 179

. Emergency Mosquito Vector Control by the City of Winnipeg - R. Ellis 180 - 182

. The Role of the Manitoba Emegency Measures Organiation in the 1981 Health Emergency - N. Reilander 183 - 184

. Emergency Mosquito Vector Control Operations - R. Ellis 185 - 191

. Drift and Deposition During Aerial Spraying of Baygon Over Winnipeg, August 1981 - G. B. Atkinson 192 - 208

. Effectiveness of the Emergency Mosquito Vector Control Operations - R. Ellis and R. Brust 209 - 222

CHAPTER VIII Safety Evaluation of Insecticides 223

. An Explanation of the Pre-registration Safety Evaluation of Insecticides Registered in Canada - J. Hollebone 224 - 230

. Monitoring of Pesticide Effects on Humans by Cholinesterase Testing - R. Hoare 231 - 236

. Health Considerations in the use of Insecticides in the Prevention of Western Equine Encephalitis - P. Warner 237 - 242

. The Effects of the 1981 Manitoba Emergency Mosquito Control Program on Honey Bees - D. Dixon and B. Fingler 243 - 247

. Analysis of Water Supplies Following the Aerial Spraying of Baygon - K. Plews and T. Youmans 248 - 250

. A Report on Mosquito Control Programs in Manitoba - Clean Environment Commission - E. Eagleton 251 - 254

CHAPTER IX Human Protection 255

. The Role of Personal Protection in the Prevention of Western Equine Encephalitis - D. Luckhurst, J. Eadie 256 - 258

. The Case for Control of Western Equine Encephalitis in Humans Through Vaccination - J. Fox 259 - 263

CHAPTER X Comments Made at the 1982 Western Equine Encephalitis Workshop on Manitoba's Arbovirus Surveillance and Control Programs 264

A. Invited Panelists - Western Equine Encephalitis Workshop:
- B. Francy 265 - 271
- T. Monath 272 - 275
- C. Schaefer 276 - 278
- L. Spence 279 - 280

B. Experts - Invited Comments
- H. Artsob 281
- J. Hollebone 282
- D. McLean 283
- A. Ronald 284 - 286
- G. Surgeonner 287
- B. Taylor 288 - 289
- J. Waters 290 - 291

CHAPTER XI Recommendations 292

. Research Recommendations for Future Surveillance and Control Programs on Western Equine Encephalitis in Manitoba - R. Brust 293 - 296

CHAPTER I:

Introduction

Brief Historical Review of Western Equine Encephalitis

R.A. Brust, B.S.A., M.Sc., Ph.D., F.E.S.C.

Department of Entomology, University of Manitoba
Winnipeg, Manitoba R3T 2N2

Western Equine Encephalitis (WEE) virus was first isolated in California, U.S.A., in 1930 (Meyer et al., 1931) and in Saskatchewan, Canada, in 1935 (Fulton, 1938). The virus attacks the central nervous system, and causes acute, febrile illness in equines and humans, and in its most severe form, causes inflammation and injury of the meninges, brain, spinal cord and death.

In Canada and the U.S.A., large epidemics and epizootics of WEE occurred in the prairie provinces and in the north central states in 1941, and in the Central Valley of California in 1952 (Donovan and Bowman, 1942 a, b; Jackson, 1942; Reeves and Hammon, 1962). There have been several epidemics since that time in the western United States and in Texas in 1958 and 1963-1966 (Anonymous, 1978).

In Canada, there have been 12 separate epidemics of WEE in humans since 1938, affecting Ontario, Manitoba, Saskatchewan, Alberta and British Columbia (Artsob and Spence, 1979). The disease has also been reported from Quebec, where WEE virus was serologically diagnosed to be the cause of illness in an infant (Pavilanus et al., 1957).

There have been more than 20 separate epizootics of WEE in equines since 1935 affecting the 4 western provinces of Canada (Artsob and Spence, 1979), and northwestern Ontario (Mitchell and Walker, 1941; Jackson, 1942; Lillie et al., 1976; Surgeoner et al., 1982).

Western Equine Encephalitis virus is generally believed to be maintained in a primary enzootic cycle involving wild birds and culicine mosquitoes (Holden et al., 1973). Equines and humans are "accidental" victims of this cycle when they are fed upon by an infected mosquito. Humans and horses play no role in the maintenance of this virus in nature.

The role of WEE virus in causing human illness in Canada was first recognized in 1938, when 27 and 29 cases were reported from Manitoba and Saskatchewan, respectively (Donovan and Bowman, 1942a, b; Gareau, 1941). The largest epidemic occurred in 1941, when 509 human cases and 78 deaths were reported from Manitoba (Jackson, 1942); 543 human cases and 44 deaths in Saskatchewan (Davidson, 1942); 42 human cases and 8 deaths from Alberta (McGugan, 1942).

Extensive follow-up studies were conducted on humans involved in the 1941 epidemic: i.e., clinical findings were reported by Adamson and Dubo (1942 a, b); infections in infants were studied by Medovy (1942, 1976); pathology of fatal cases was reported by Quong (1942); the isolation of WEE virus from cerebral spinal fluid was reported by Chown and Norris (1942) and Gwatkin and Moynihan (1943). Mitchell and Pullin (1943) reported that 18.95% of a sample of 1013 Manitoba residents had neutralizing antibodies following the 1941 outbreak.

In western Canada, WEE is a late summer disease (July-September). It occurs in horses 2-3 weeks before human cases are reported. The delay in humans is primarily due to the longer incubation period required to cause illness.

References Cited

Adamson, J.D. and S. Dubo, 1942a. Clinical findings in encephalitis (Western equine). Can. Med. Assoc. J. 46: 530-537.

Adamson, J.D. and S. Dubo, 1942b. Clinical findings in encephalitis (Western type). Can. Public Hlth J. 33: 288-300.

Anonymous, 1978. Control of Western Equine Encephalitis. Vector Topics No. 3. U.S. Dept. Hlth, Educ. and Welfare.

Artsob, H. and L. Spence, 1979. Arboviruses in Canada, p. 39-65. In Arctic and Tropical Arboviruses. E. Kurstak (ed.). Academic Press.

Chown, B. and M. Norris, 1942. Virus of western equine encephalitis in the spinal fluid. J. Am. Med. Assoc. 120: 116-117.

Davidson, R.O., 1942. Encephalomyelitis in Saskatchewan, 1941. (Preliminary report). Can. Publ. Hlth J. 33: 388-398.

Donovan, C.R. and M. Bowman, 1942a. Epidemiology of encephalitis, western equine type, Manitoba, 1941. Can. Med. Assoc. J. 46: 525-530, 530-537.

Donovan, C.R. and M. Bowman, 1942b. Some epidemiologic features of poliomyelitis and encephalitis, Manitoba, 1941. Can. Publ. Hlth J. 33: 246-251.

Fulton, J.S. 1938. A report of two outbreaks of equine encephalomyelitis in Saskatchewan. Can. J. Com. Med. II(2): 39-46.

Gareau, U. 1941. Clinical aspects of an epidemic of human encephalomyelitis in Saskatchewan in 1938. Can. J. Publ. Hlth 32(1): 1-5.

Gwatkin, R. and I.W. Moynihan, 1943. Recovery of equine encephalitis virus (western type) from human spinal fluid in Alberta. Can. J. Publ. Hlth 34: 166-170.

Holden, P., R.O. Hayes, C.J. Mithcell, D.B. Francy, J.S. Lazuick, and T.B. Hughes, 1973. House Sparrows, Passer domesticus (L.) as hosts of Arboviruses in Hale County, Texas. 1. Field studies, 1965-1969. Am. J. Trop. Med. Hyg. 22: 244-253.

Jackson, F.W., 1942. Encephalitis, Can. Med. Assoc. J. 46:364-356.

Lillie, L.E., F.C. Wong and R.A. Drysdale, 1976. IV. Equine epizootic of Western encephalomyelitis in Manitoba - 1975. Can. J. Publ. Hlth 67 (Suppl. I):21-27.

McGugan, A.C., 1942. Equine encephalomyelitis (western type) in humans in Alberta, 1941. Can. Publ. Hlth 33: 148-151.

Medovy, H., 1942. Western equine encephalomyelitis in infants. Can. Publ. Hlth J. 33: 307-312.

Medovy, H., 1976. II. The history of western encephalomyelitis in Manitoba. Can. J. Publ. Hlth 67 (Suppl. I):13-14.

Meyer, K.F., C.M. Haring and B. Howitt, 1931. The etiology of epizootic encelphalomyelitis in horses in the San Joaquin Valley, 1930. Science 74:227-228.

Mitchell, C.A. and J.W. Pullin, 1943. Examination of sera from persons in Manitoba, Ontario and Quebec for neutralizing antibodies (western type) of encephalomyelitis. Can. J. Publ. Hlth 34: 419-420.

Mitchell, C.A. and R.V.L. Walker, 1941. Studies in equine encephalomyelitis.- Susceptibility of some mammals and birds. Can. J. Comp. Med. 5: 314-319.

Pavilanus, V. et al., 1957. Western equine encephelomyelitis: report of a case in Montreal. Can. Med. Assoc. J. 77: 128-130.

Quong, T.L., 1942. The pathology of western equine encephalomyelitis. Can. Publ. Hlth J. 33: 300-306.

Reeves, W.C. and W. McD. Hammon, 1962. Epidemiology of the arthropod-borne viral encephalitides in Kern County, California 1943-1952. Univ. Calif. Press, Berkeley. 257 pp.

Surgeoner, G.O., 1982.

Surgeoner, G.O., W.E. Ralley and M.S. Mahdy, 1982. Investigation for western equine encephalitis activity in Northwestern Ontario, 1981. In Press.

Manitoba Arbovirus Surveillance Committee: Historical Review, Terms of Reference and Structure

L. Sekla, M.B.B.Ch., Ph.D.

Manitoba Health Services Commission
Cadham Provincial Laboratory 750 William Avenue
Winnipeg, Manitoba R3C 3Y1

Since the early thirties, summer epizootics and epidemics of Western Equine Encephalitis (WEE), although sporadic, were severe enough to trigger a state of vigil in Manitoba. During the 1941 epidemic, it became clear that a coordinated effort was necessary to pool all available information on human and equine cases. After the epidemic, the need to consolidate all the data was identified and resulted in a monograph sponsored by Manitoba's Department of Health, (Jackson _et al_ 1942). However, from 1947 until 1974, no WEE outbreaks (defined as more than two cases per summer) were reported and the need for surveillance became less and less pressing.

In 1975, when an epidemic of WEE became imminent, the 1941 experience was quickly reviewed and specialists brough together to monitor the situation. By then, the variety of disciplines involved had grown tremendously compared to the forties. The 1975 _ad hoc_ multidisciplinary committee was comprised of entomologists, meteorologists, epidemiologists, veterinarians, environmentalists, laboratorians and public information officers. This committee documented the 1975 WEE outbreak, (Sekla _et al_ 1976). The committee also recommended the formation of a permanent, multidisciplinary body to conduct an annual surveillance program in epidemic and non-epidemic years. This recommendation was approved by the Minister of Health and implemented immediately. The Manitoba Arbovirus Surveillance Committee (MASC) was formed with representation from the University of Manitoba, the City of Winnipeg, the Federal and Provincial Governments; MASC has been meeting regularly since 1976 and has successfully predicted the 1977 and 1981 WEE outbreaks and the non-outbreak years inbetween.

MASC's terms of reference "are to collect and assess evidence of WEE as it becomes available and as necessary, to advise the Minister of Health of the risk to the population and what remedial measures could be taken". When an epidemic of WEE appears to be imminent, MASC informs the Minister of Health who in turn consults with the Provincial Cabinet, convenes an Emergency Task Force, and if deemed necessary, declares a health emergency.

References Cited

Jackson, F.W., _et al_, 1942. "Symposium: poliomyelitis and encephalitis Manitoba 1941". Can. J. Public Health. 33:241-314.

Sekla, L., et al, 1976. "Western Encephalomyelitis". Can. J. Public Health. 67 (Suppl.):1-75.

Manitoba Arbovirus Surveillance Committee: Description of the Surveillance Program

L. Sekla M.B.B.Ch., Ph.D.

Manitoba Health Services Commission
Cadham Provincial Laboratory 750 William Avenue
Winnipeg, Manitoba R3C 3Y1

To achieve its goals, the Manitoba Arbovirus Surveillance Committee (MASC), in accordance with its terms of reference, developed a surveillance program consisting of the following 3 levels: i.e.,

1. Data Collection:

This is an elaborate process carried out by a number of agencies representing a variety of disciplines. It involves the following:

1. The collection, counting and identification of mosquitoes.
2. The isolation of arboviruses from live-trapped mosquitoes.
3. The placing, bleeding and serologically testing of sentinel chicken flocks.
4. Monitoring the clinical picture, serological findings, and immunization status of sick horses.
5. Active surveillance of humans with symptomatology compatible with an arboviral infection.

Every year the Provincial Epidemiologist requests funds to cover the expenses of the data collection and the salaries of the summer students. The number of geographic locations in which sentinel chicken flocks are placed and from which mosquitoes are collected vary depending on annual budget allocations.

2. Data Analyses

The data collected are recorded and analyzed by each agency before submission to MASC which integrates the information received from all disciplines and attempts to determine the risk of the epidemic. For this purpose a set of predictive criteria has been developed and refined over the years and will be presented in the chapters on weather conditions, Culex tarsalis populations, arbovirus isolations from mosquitoes, seroconversions in sentinel chicken flocks and number of reported equine cases.

3. Dissemination of Information

This step is necessary to keep senior government officials, the medical and veterinary professions and the public appraised of the potential for arboviral infection. An exchange of information with neighbouring jurisdictions is also an essential role of MASC.

Health Emergency - The Decision Making Process

Eadie, J.A. M.B., Ch.B., D.P.H.

Preventive Medical Services, 831 Portage Avenue,
WINNIPEG, Manitoba R3G 0N6

Abstract

The control of an epidemic of Western Equine Encephalitis (WEE) cannot await the diagnosis and reporting of human cases. The entire process of monitoring, assessment, planning and delivering the counter measures must be completed before human cases occur. This is a unique situation in Communicable Disease Control. This article outlines the sequential steps taken in this process.

The Manitoba Arbovirus Surveillance Committee (MASC) is appointed by the Minister of Health to advise him of the risk of an epidemic of Arbovirus infection with particular emphasis on Western Equine Encephalitis (WEE), in Manitoba.

Due to the fact that there had been no epidemic since 1977, the funds voted for the program were reduced. This meant that the program was only able to operate 5 live traps at Glenlea, Charleswood Sewage Lagoons and Oak Hammond Marsh in the vicinity of Winnipeg and in Portage La Prairie 80 Km and Brandon 215 Km west of Winnipeg, instead of the recommended 15. The City of Winnipeg maintained their full complement of New Jersey Light Traps in and around the City.

The fall of 1980 had been relatively dry and the winter of 1980-81 had been relatively warm. These factors suggested that there would be few mosquitoes in the summer of 1981. It appeared that this early assessment was to be fulfilled as there was no indication of impending danger until the week of 20 July.

A regular meeting of MASC was scheduled for Thursday, 23 July. During 20-21 July reports were received from monitoring sources which changed the situation dramatically. It was presented to the MASC at its meeting of 23 July.

1. The McLintock Formula, (McLintock, 1948) was fulfilled.
2. Mosquito numbers and the percentage of Culex tarsalis were equivalent to the epidemic years 1975 and 1977.
3. 4 Mosquito Pools were positive for WEE virus.
4. 7 confirmed horse cases of WEE were reported. This greatly exceeded the relevant threshold of 6 suspect horse cases/week.
5. The majority of chicken flocks sero-converted to WEE at significant titres, equivalent to those observed in the epidemic years, 1975 and 1977.

After considerable discussion the Committee made the decision that THERE WAS A RISK OF AN EPIDEMIC OF WEE IN MANITOBA IN 1981.

The following action was proposed:-

1) The Minister of Health was to be immediately warned of this risk, its' URGENCY and the need to consider and prepare for ACTION at the Provincial level.

2) The City of Winnipeg was to re-start fogging residential areas, which had just been completed and to larvicide to a 25 Km radius.

3) All Manitobans were to be warned of the risk and to avoid exposure to mosquitoes.

4) Rural communities were to be advised to fog, if they had equipment.

The warning was issued as a news release dated 23 July by the Province of Manitoba News Service.

The period of 24-26 July was spent planning and implementing a Province-wide program. Chemicals and a spray-equipped DC6 were provisionally ordered; legislative requirements were prepared.

On 27 July, the initial meeting of the Emergency Measures Organization (EMO) Task Force was held. MASC was amongst numerous areas of responsibility represented on this Task Force. EMO assumed the responsibility of coordinating the emergency action from that time on. Orders for chemicals and the plane were confirmed. The declaration of a health emergency by the Minister of Health was prepared; it was to be signed 30 July and "filed" 31 July in the Manitoba Gazette.

An emergency meeting of Cabinet was held to discuss the health emergency. Funds required were approved.

Dr. Roy Ellis was seconded from the City of Winnipeg to the Province of Manitoba and placed in charge of Spraying Operations.

A letter was sent to all Manitoba Physicians by the Provincial Epidemiologist outlining the situation, describing the clinical picture, diagnostic procedures and preventive measures.

By 30 July there were 16 confirmed horse cases.

A major decision to be made by the Committees was the "target" for spraying. It was recognized that mosquitoes were more numerous in rural areas. After careful consideration of population, topography, entomology, weather, distribution of horse cases, location of virus identifications and other risk factors - it was agreed to focus on spraying population centres with emphasis on size, since fundamentally it was people who we were trying to protect. Even in the largest cities there were still considerable areas of rural type land intruding into populated areas. Ultimately 21 communities were sprayed, with a total population of 680,806 and a minimum population of approximately 1,800 - Fig. 1.

Questions were raised about the legal status of the licencing of Baygon (propoxur). Confirmation was obtained from appropriate Government authorities that it was approved for use as a Mosquito insecticide by the Governments of Canada as well as the U.S.A.

On 4 August - spraying began.

On 5 August, on 1½ hours notice, a Court Injunction had to be defended. It was submitted by a group of citizens concerned about environmental damage. The Court decided that the Province had the legal right to spray and to use Baygon. It was not asked to decide on the merits of the need to spray. Spraying resumed the following day.

The media and the "environmentalists" repeatedly put forward an image that there was a "Baygon Problem." The Government and the Department of Health recognized that the problem was one of WEE and that Baygon was one part of the solution.

WARNINGS were issued constantly and repeatedly to Manitobans through the Media, Press Conferences and News Releases to USE PERSONAL ANTI-MOSQUITO PROTECTION, paying special attention to young children. This was a MAJOR component of our program, though it tended to get lost in the "Baygon controversy."

On 10 August confirmed horse cases equalled the grand total of horse cases for the 1977 Epidemic year.

On 13 August the first human WEE case was diagnosed - the date of onset had been 16 July and infection presumably occurred between 1 and 11 July. It should be noted that all MAJOR decisions had to be and were made long before the diagnosis of this first case, on other factors than the incidence of human cases. The final total of human cases of WEE was 25, with 2 deaths, caused or precipitated by the disease.

After mid August a complex question was before the committees:-

When would "hibernation" reduce the number of mosquitoes feeding to the point when spraying would no longer be required? The continued warm humid weather kept mosquitoes feeding until the spraying program was completed. Furthermore, there was also an increase in activity by Culiseta inornata expected with cooler weather. (It is possible that this may have been the source of the human case in Parklands Region, presumed to have been infected in September).

It is impossible to determine the effectiveness of the combined program of:-

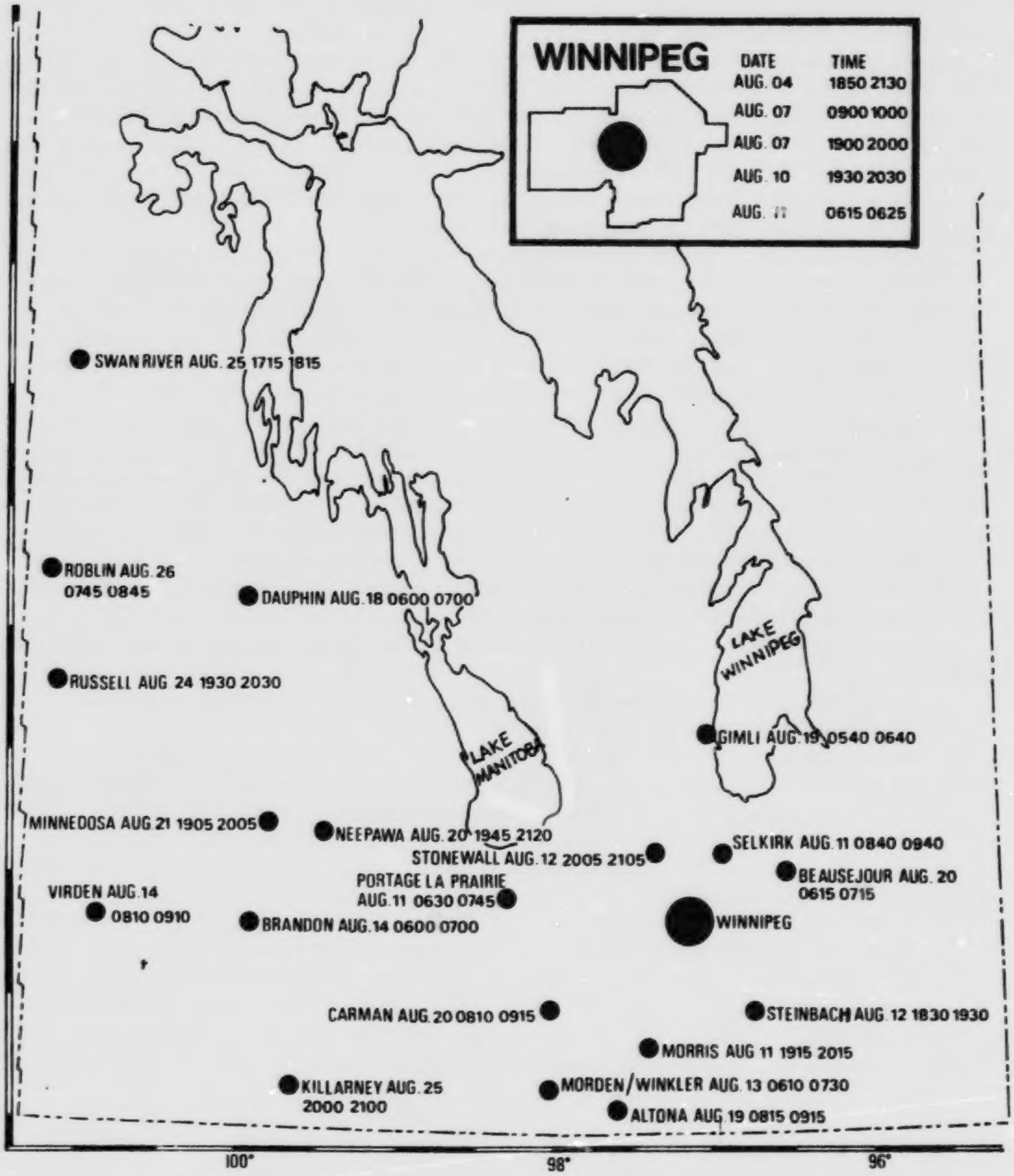
1. Encouraging personal protection against mosquito exposure.
2. Spraying population centres.

However, the relatively few cases and deaths in the human population compared to the widespread distribution of horse cases and virus isolations tends to suggest the right decisions were made.

Reference Cited

McLintock, J., 1948. "Report of the Virus Laboratories for 1947." Manitoba Department of Health and Public Welfare, Provincial Laboratories, 161-168.

Figure 1. - Communities Sprayed with Propoxur 1981.


WINNIPEG
DATE
TIME
AUG. 04 1850 2130
AUG. 07 0900 1000
AUG. 07 1900 2000
AUG. 10 1930 2030
AUG. 11 0615 0625
SWAN RIVER AUG. 25 1715 1815
ROBLIN AUG. 26 0745 0845
DAUPHIN AUG. 18 0600 0700
RUSSELL AUG 24 1930 2030
LAKE WINNIPEG
GIMLI AUG. 19 0540 0640
LAKE MANITOBA
MINNEDOSA AUG 21 1905 2005
NEEPAWA AUG. 20 1945 2120
STONEWALL AUG. 12 2005 2105
SELKIRK AUG. 11 0840 0940
BEAUSEJOUR AUG. 20 0615 0715
PORTAGE LA PRAIRIE AUG. 11 0630 0745
VIRDEN AUG. 14 0810 0910
BRANDON AUG. 14 0600 0700
WINNIPEG
CARMAN AUG. 20 0810 0915
STEINBACH AUG. 12 1830 1930
MORRIS AUG 11 1915 2015
KILLARNEY AUG. 25 2000 2100
MORDEN/WINKLER AUG. 13 0610 0730
ALTONA AUG. 19 0815 0915
100°
98°
96°

The Emergency Public Information Program

N.R. Donogh
Director

Manitoba Information Services Branch
Legislative Building
Winnipeg, Manitoba

Abstract

A brief description of the public information program carried out by the Manitoba Information Services Branch is given including media releases, press conferences and the operation of a news centre.

The public information program associated with the 1981 Western Equine Encephalitis (WEE) health emergency was designed to advise citizens of the health risk, the protective measures they could take, and the steps being taken by the Province.

In the pre-emergency period, beginning in late May, the information program explained monitoring procedures, the role of the Manitoba Arbovirus Surveillance Committee (MASC), and the steps farmers should take in the vaccination of horses.

As viral activity among sentinel chicken flocks and horse cases increased the news release dealt with the increasing threat of WEE and advised citizens on precautions they should take themselves and for their children.

Internally, the Information Services Branch was represented at meetings of MASC and the Emergency Committee of Cabinet. Procedures were also put in place for the operation of a special news centre during the emergency.

Once the emergency was declared on July 30, the Cabinet Committee held a news conference outlining the spraying program, the decision to use Baygon MOS, and special protective procedures to be taken by beekeepers and those with respiratory illnesses. From that point on, information centered on time, place and nature of the spraying program, changes in viral activity, and the incidence of the disease among humans and horses. The newsroom operated from 5 a.m. daily (when final decisions were made concerning morning spraying) to midnight (when the Manitoba Emergency Measures Organization or EMO operations office closed). Each day the Health Minister and Government Services Minister (responsible for EMO) held news briefings to update the media. Data was assembled and statements prepared by Information Services. The Branch kept in constant touch with the media, providing direct voice reports every 3 hours to the electronic media and compiling written reports twice daily for the print media.

A major issue concerned the use of Baygon, a controversy led by a number of the scientific community. As well, some news stories based on "label" warnings on Baygon, rather than on the use of highly-diffused Baygon MOS in spray form, led to considerable public concern. Stories of "chemical bombs", of an attempted court injunction against use of Baygon spray, of the alleged death of a child as a result of Baygon spraying during a previous emergency in Windsor, and of concerns expressed by the Canadian Wildlife Service all added to a situation where many citizens were more concerned about Baygon spray than about the effects of WEE itself.

To help offset this, respected scientists and a respirologist briefed the media at one of the daily conferences early in the emergency and further reminders were issued on the effects of earlier outbreaks of the disease. As the emergency continued, people generally were more supportive of the spraying program - particularilly after the first human case was reported - and a number of communities outside the threatened area were also pressing to be included in the spraying program. There appeared to be general acceptance of the emergency measures that were undertaken.

CHAPTER II:

Mosquito Data

Population Dynamics of Culex tarsalis Coquillett in Manitoba

R.A. Brust, B.S.A., M.Sc., Ph.D. Entomology, F.E.S.C.

Department of Entomology, University of Manitoba,
Winnipeg, Manitoba R3T 2N2

ABSTRACT

Sentinel flock trap collections of mosquitoes over 6 years (1976-81) indicate that the numbers and percentage (in comparison to other species) of Culex tarsalis are correlated with the 2 years (1977, 1981) out of 6, when human and equine outbreaks of Western Equine Encephalitis (WEE) occurred ($r = 0.76$ for numbers, and $r = 0.89$ for percentage). The parameters > 20 C. tarsalis females/trap/week during late June or early July and > 25% C. tarsalis females in flock trap collections during this period are criteria which might be used in future years to predict outbreak population levels of C. tarsalis.

Culiseta inornata (Williston) and Aedes vexans (Meigen) are unlikely to be involved in WEE virus transmission to equines and humans during July or August, because females are infected at very low levels and their population peaks do not coincide with transmission peaks for WEE. They may be involved in amplifying the virus in spring.

Although Western Equine Encephalitis (WEE) virus has been isolated from a variety of mosquito species in nature (Reeves and Hammon, 1962; McLintock et al., 1970; Sekla et al., 1980), Culex tarsalis Coquillett is considered to be the major vector of this virus wherever the disease occurs.

This mosquito readily takes blood from avian, equine and human hosts and hence acquires the virus from the avian (summer) reservoir and transmits it to humans and equines. Once infected, a female C. tarsalis is able to transmit the virus throughout her life (Henderson et al., 1979). A female mosquito that acquires virus in a bloodmeal a few days after emergence, may live another 1-4 weeks. She could take 1-4 more blood meals (some from equines or humans) and infect an equal number of hosts with WEE virus. She may infect more if she bloodfeeds on more than 1 animal during each feeding cycle.

In order to determine when and which mosquito species were infected with WEE virus in Manitoba, mosquito adults were live-trapped at various sites throughout the province. Virus isolations were carried out at the Cadham Provincial Laboratory (Sekla and Stackiw, 1982).

The main objectives of this paper are to show how collections of female C. tarsalis in Manitoba varied over a period of 6 years (1976-1981) and to show how human and equine outbreaks of WEE were related to C. tarsalis collections.

METHODS

A light and suction fan trap (Fig. 1), similar to one used by McLintock (1946), was used to collect mosquitoes at 5 rural sites around Manitoba (3 near the major centres of Winnipeg, Portage la Prairie and Brandon, Fig. 2).

A sentinel chicken flock trap (Fig. 3), similar to that of Rainey et al. (1962), was used to collect female mosquitoes attracted to avian hosts. This was operated at the above locations, 50 to 100 m from the light trap.

Both the flock and the light trap were operated continuously throughout the summer (June, July, August) and emptied daily (Monday-Friday) during 1976 and 1977, but only three times weekly (Monday, Wednesday, Friday) during 1978-1981. Live mosquitoes were transported in insulated coolers (5-10°C) to the laboratory, where they were sorted to species. Only live mosquitoes were processed for virus isolation (Sekla and Stackiw, 1983).

RESULTS

The number of C. tarsalis and percentage of all species collected from sentinel flock traps is shown (Table 1). The two WEE epidemic years (1977, 1981) differ substantially from the other 4 years. The numbers and percentage of C. tarsalis in the collection is greatest for those years.

In 1977, the mosquito season began early. C. tarsalis was present in significant numbers by 8 June (Table 1). The numbers remained high until the middle of August. The seasonal collection of all species, including C. tarsalis, was greatest in 1977 when compared to the other 5 years. The seasonal percentage of C. tarsalis, in relation to all mosquitoes captured in the flock traps, was 52.5%. The percentage of C. tarsalis during July, 1977, was 63% of all mosquitoes captured.

During 1981, C. tarsalis females were not captured in significant numbers until 30 June (Table 1). The collections increased very markedly in the flock trap, parallelling the 1977 numbers and percentage of C. tarsalis. The seasonal percentage of C. tarsalis was 48.2 and the July percentage was 62.

The flock trap collections, on a seasonal basis, indicate a strong correlation between the number of C. tarsalis captured during the 2 WEE epidemic years compared to the 4 non-epidemic years (r = 0.76). The percentage of C. tarsalis collected during the 2 WEE epidemic years is correlated at an even higher level (r = 0.89). The month of July shows the greatest contrast in numbers captured, and percentage C. tarsalis, during WEE epidemic and non-epidemic years (Table 1). The increase in both numbers and percentage of C. tarsalis, during late June and early July, is characteristic for WEE epidemic years and may be of predictive value. The first WEE equine case occurred on 26 June 1977 and 16 July 1981 (Figs. 4, 5). The numbers and percentage of C. tarsalis increased significantly 1-2 weeks prior to the first equine case in both years.

During WEE epidemic years, the light trap collections of *C. tarsalis* (Table 2) show that higher numbers of *C. tarsalis* were present on a seasonal basis. A greater seasonal percentage of *C. tarsalis* was not observed. During July, both numbers and percentage of *C. tarsalis* were greatest for WEE epidemic years.

The numbers of *C. tarsalis* collected on a weekly basis in flock traps are also much greater for WEE epidemic years than for non-epidemic years (Figs. 6, 7). Flock trap collections indicate a late June - early July rise in numbers of *C. tarsalis* during WEE epidemic years, 1-2 weeks earlier than light trap collections (Tables 1, 2; Figs. 6, 7).

DISCUSSION

Mosquito collections indicate that flock traps detect the rise in *C. tarsalis* populations 1-2 weeks earlier than light traps. This is a critical factor in predicting potential epidemics. It appears that *C. tarsalis* must begin a major population increase before 15 July in Manitoba if it is going to produce seasonal population totals similar to those occuring during epidemic years. Therefore, a careful analysis of *C. tarsalis* collections each week during this time is critical. During epidemic years, the flock trap collections exceeded 75 *C. tarsalis* per trap/week during the weeks of 8 and 15 July and exceeded 38% of the total mosquitoes collected. During non-epidemic years, the collection was below 20/trap/week and below 24% of the total mosquitoes collected. The parameters, > 20 *C. tarsalis* per trap per week and > 25% *C. tarsalis* in flock trap collections, might be used as a guide to predict future epidemics in Manitoba, assuming coincident high WEE virus activity. These parameters can later be adjusted as more seasons are monitored.

Acknowledgements

I am grateful to the many summer students who assisted in gathering the data, the Manitoba Youth Secretariat, and the Manitoba Department of Health for funding, to Dr. M.M. Chance for assisting me in supervising the students.

References Cited

Henderson, L.P., R.A. Brust and F.C. Wong, 1979. Biological transmission of western encephalomyelitis virus by *Culex tarsalis* Coquillett. Mosquito News 39: 385-390.

McLintock, J. 1946. Collecting and handling mosquitoes or Western Equine Encephalitis investigations in Manitoba. Can. J. Res. E. 24: 55-62.

McLintock, J., A.N. Burton, J.A. McKiel, R.R. Hall and J.G. Rempel, 1970. Known mosquito hosts of Western Encephalitis virus in Saskatchewan. J. Med. Ent. 7: 446-454.

Rainey, M.B., G.V. Warren, A.D. Hess and J.S. Blackmore, 1962. A sentinel chicken shed and mosquito trap for use in encephalitis field studies. Mosquito News 22: 337-342.

Reeves, W.G. and W. McD. Hammon, 1962. Epidemiology of the arthropod-borne viral encephalitides in Kern County, California: 1943-1952. Univ. Calif. Press, Berkeley. 257 pp.

Sekla, L. and W. Stackiw, 1982. Arbovirus isolation and serology in Manitoba. (In press).

Sekla, L., W. Stackiw and R.A. Brust, 1980. Arbovirus isolations from mosquitoes in Manitoba. Mosquito News 40: 377-380.

Table 1. Number of *Culex tarsalis* and percent of all species collected in flock traps (5) in Manitoba during 1976-81

Month	Dates	1976		1977		1978		1979		1980		1981	
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
June	1-8	0	0	18	9	0	0	1	<1	0	0	0	0
	9-15	2	<1	21	14	0	0	0	0	0	0	1	4
	16-23	15	3	119	16	3	<1	8	1	0	0	1	1
	24-30	40	5	278	27	33	8	2	<1	2	7	14	17
July	1-8	81	20	515	38	12	4	23	3	4	11	336	69
	9-15	73	25	866	64	31	11	97	11	9	24	603	50
	16-23	233	45	1246	79	329	48	6	8	10	16	451	53
	24-31	268	63	795	70	374	53	188	54	3	50	912	78
July total:		655	40	3422	63	746	37	314	15	26	18	2302	62
August	1-8	29	44	1174	69	149	43	115	44	8	61	143	59
	9-15	75	56	207	49	73	24	42	19	0	-	57	56
	16-23	78	67	6	7	18	51	40	35	-	-	8	3
	24-31	-	-	2	3	-	-	-	-	-	-	27	4
Seasonal total:		894	23.7	5247	52.5	1022	25.7	522	13.8	36	16	2553	48.2

Table 2. Number of *Culex tarsalis* and percent of all species collected in light traps (5) in Manitoba during 1976-81

Month	Dates	1976		1977		1978		1979		1980		1981	
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
June	1-8	1	<1	156	15	0	0	1	<1	2	2	8	5
	9-15	8	3	124	20	0	0	0	0	7	3	9	2
	16-23	9	5	232	19	12	<1	6	1	6	5	23	2
	24-30	25	8	133	20	63	9	1	<1	7	6	52	5
July	1-8	22	8	156	18	32	10	35	6	5	10	100	18
	9-15	23	8	589	63	192	11	42	21	15	14	176	42
	16-23	46	27	1423	68	504	10	73	21	68	32	419	42
	24-31	65	38	1029	44	419	15	215	35	44	43	349	43
July total:		156	16	3197	51	1147	12	365	21	132	23	1044	37
August	1-8	47	40	1570	50	47	4	244	22	56	33	449	34
	9-15	33	34	327	27	121	3	160	30	47	26	204	27
	16-23	13	21	27	7	38	14	14	4	11	10	103	7
	24-31	-	-	10	4	-	-	1	-	-	-	179	7
Seasonal total:		292	13.7	5776	39.2	1428	7.2	791	12.4	268	17.6	2071	17.4

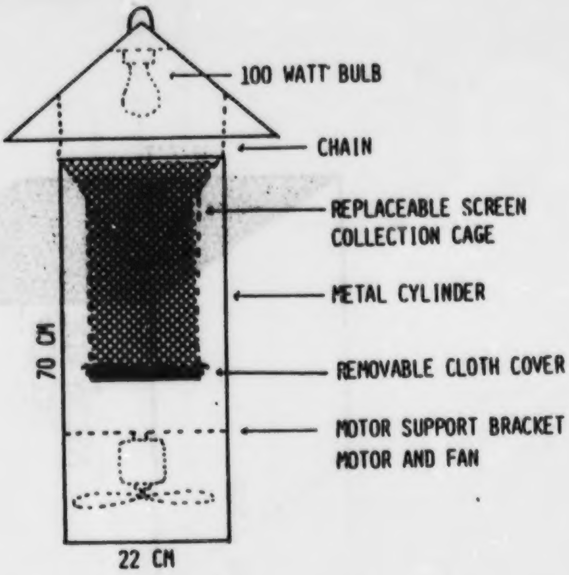

100 WATT BULB
CHAIN
REPLACEABLE SCREEN COLLECTION CAGE
METAL CYLINDER
REMOVABLE CLOTH COVER
MOTOR SUPPORT BRACKET
MOTOR AND FAN
70 CM
22 CM

FIG. 1. MOSQUITO LIGHT TRAP USED IN LIVE TRAPPING FOR W.E.E. VIRUS ISOLATION

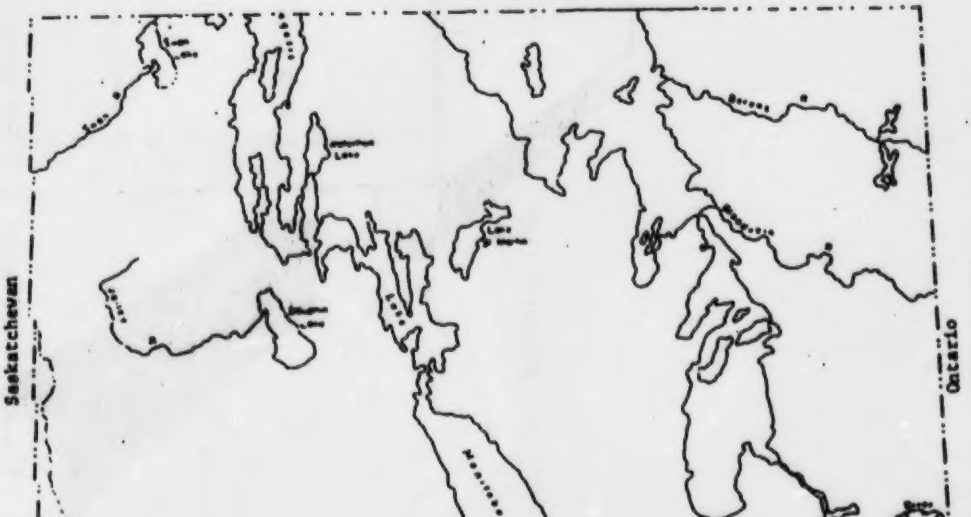

Saskatchewan
Ontario
Oak Hammock
Portage la Prairie
Brandon
Winnipeg
Glenlea

FIG. 2. FLOCK AND LIGHT TRAP LOCATIONS IN MANITOBA 1976-81.

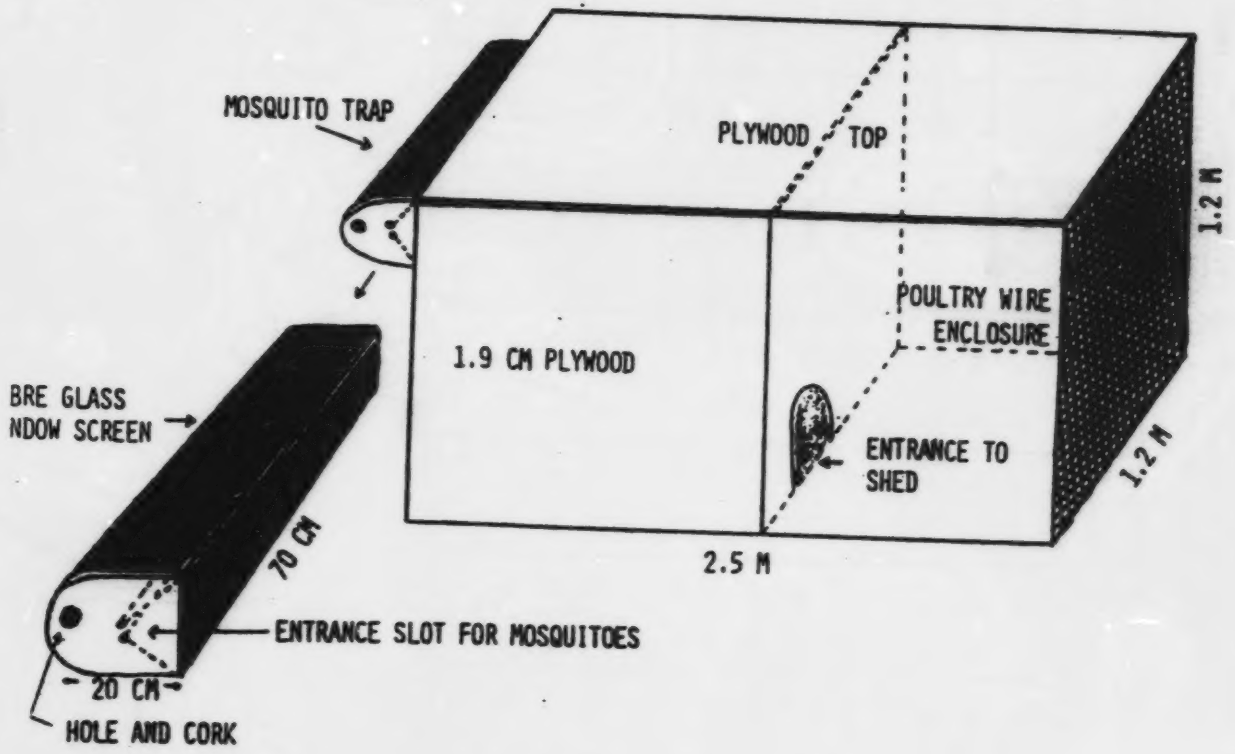

MOSQUITO TRAP
PLYWOOD TOP
1.2 M
POULTRY WIRE ENCLOSURE
1.9 CM PLYWOOD
BRE GLASS
NDOW SCREEN
ENTRANCE TO SHED
1.2 M
70 CM
2.5 M
ENTRANCE SLOT FOR MOSQUITOES
20 CM
HOLE AND CORK

FIG. 3 SENTINEL CHICKEN SHED. WIRE ENCLOSURE AND MOSQUITO FLOCK TRAP.

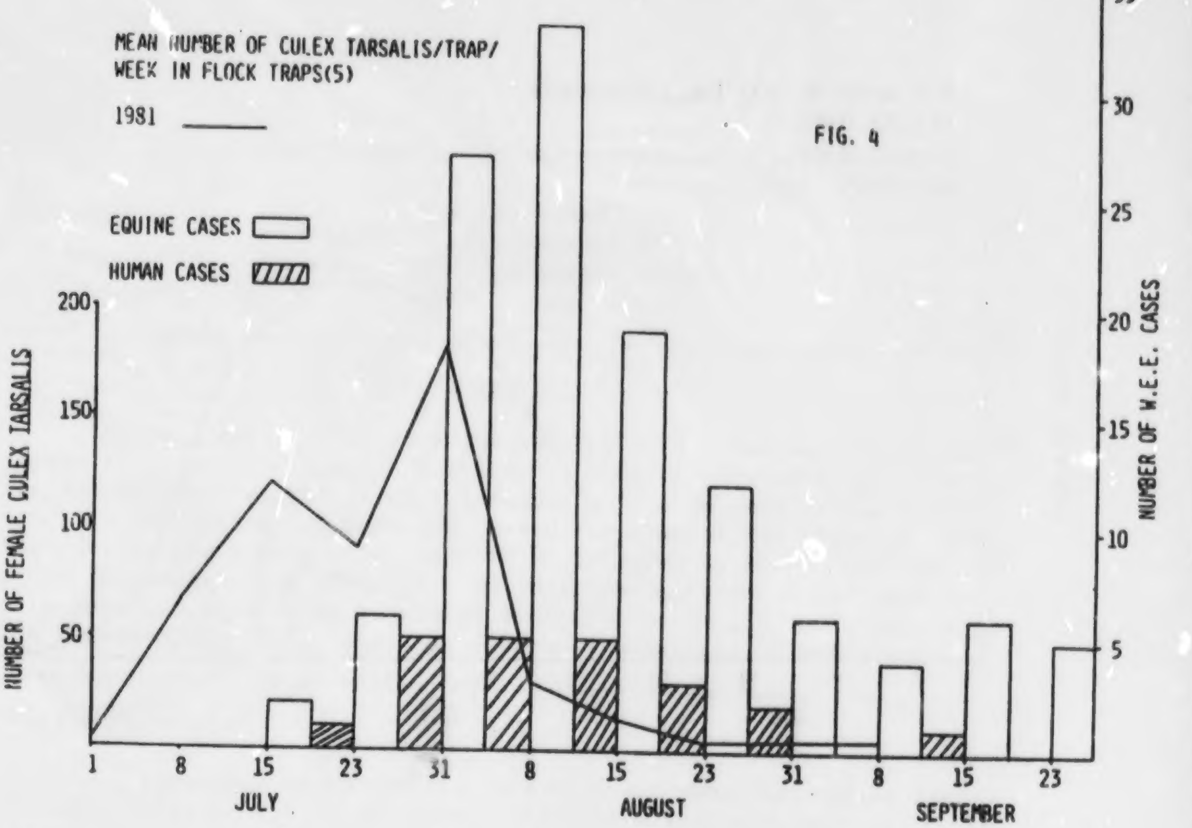

MEAN NUMBER OF CULEX TARSALIS/TRAP/
WEEK IN FLOCK TRAPS(5)
1981 ———
EQUINE CASES
HUMAN CASES
200
150
100
50
NUMBER OF FEMALE CULEX TARSALIS
35
30
25
20
15
10
5
NUMBER OF W.E.E. CASES
1
8
15
23
31
8
15
23
31
8
15
23
JULY
AUGUST
SEPTEMBER

FIG. 4

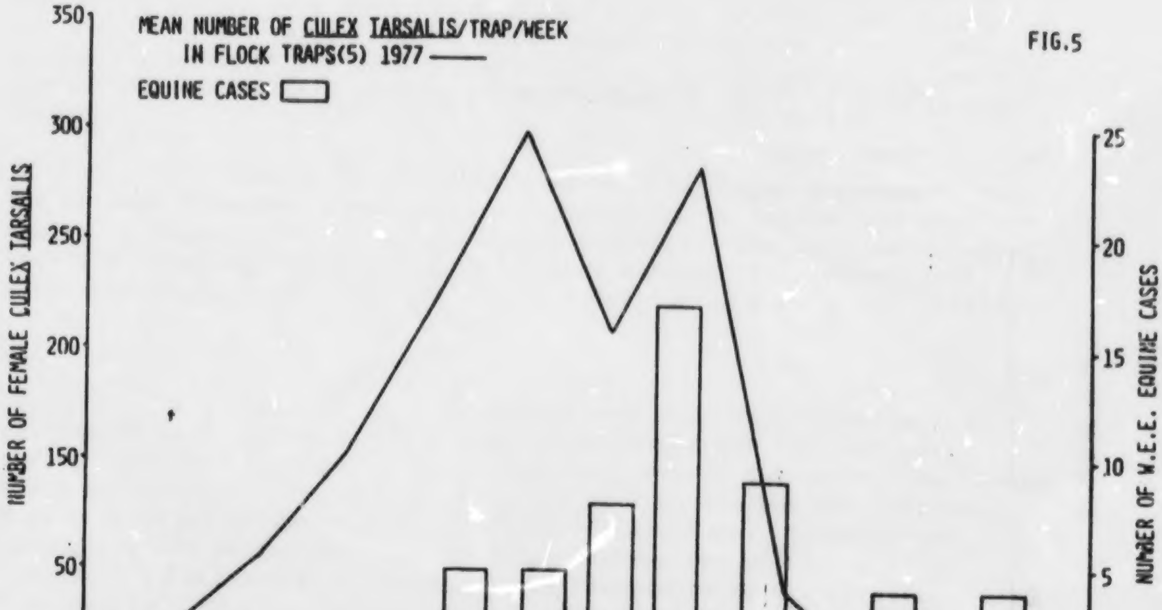
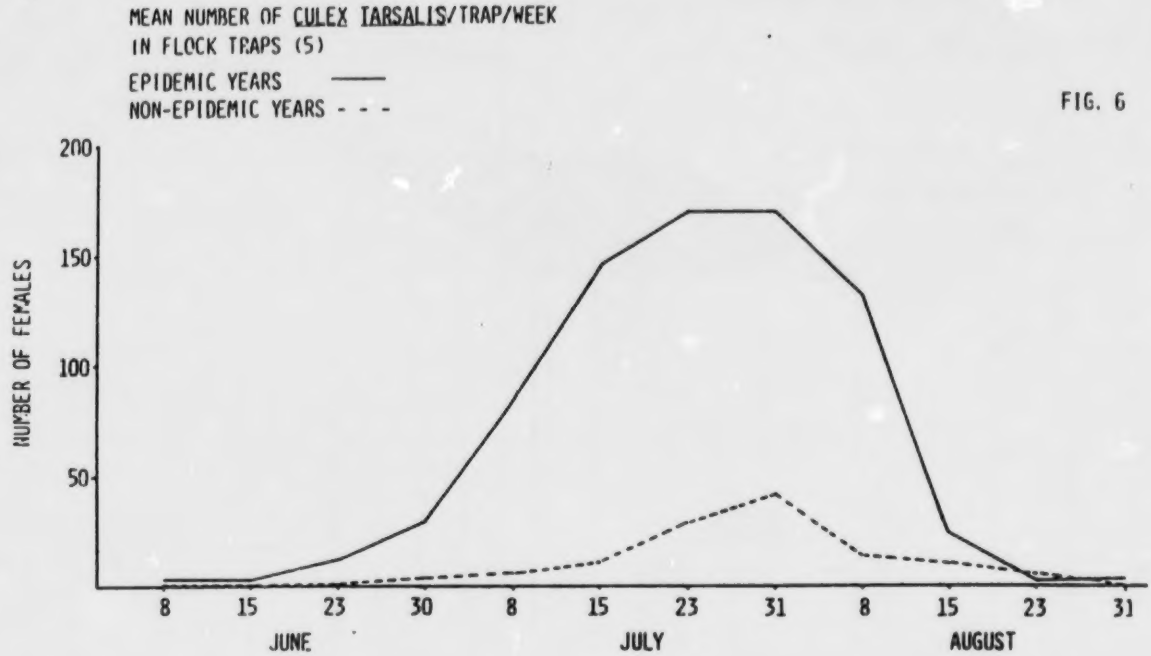

MEAN NUMBER OF CULEX TARSALIS/TRAP/WEEK
IN FLOCK TRAPS(5) 1977 ———
EQUINE CASES
350
300
250
200
150
50
NUMBER OF FEMALE CULEX TARSALIS
25
20
15
10
5
NUMBER OF W.E.E. EQUINE CASES
15
23
30
8
15
23
31
8
15
23
31
8
JUNE
JULY
AUGUST
SEPTEMBER

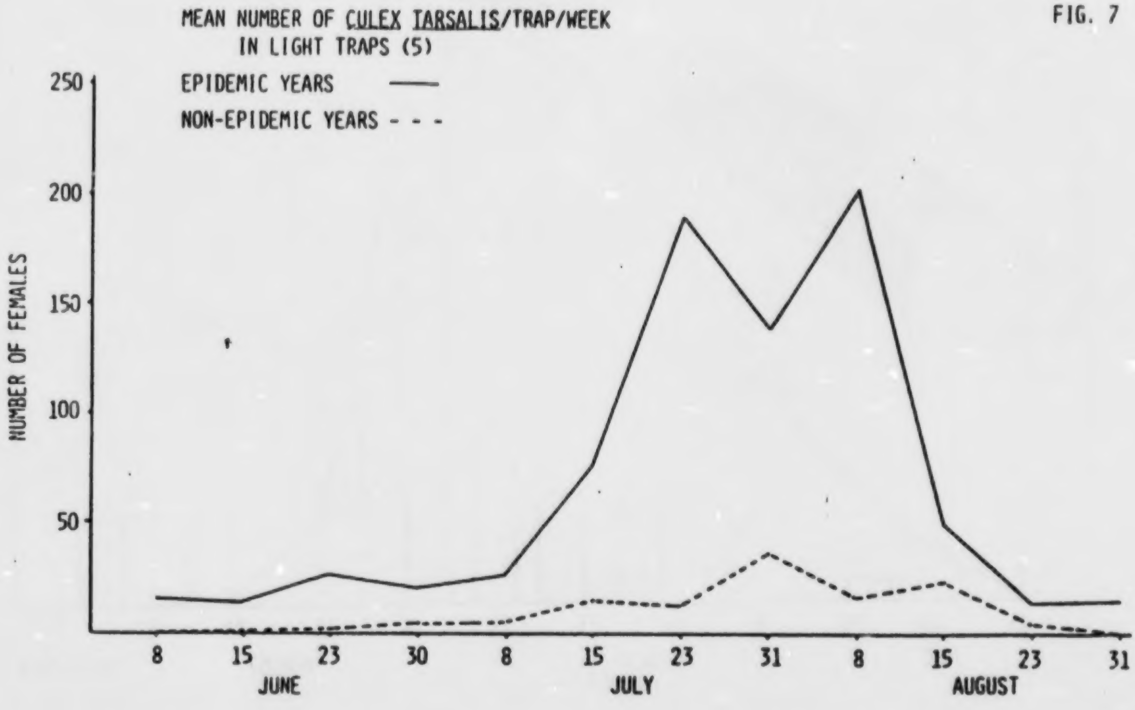
FIG.5

FIG. 6



MEAN NUMBER OF *CULEX TARSALIS*/TRAP/WEEK
IN FLOCK TRAPS (5)

FIG. 7



MEAN NUMBER OF *CULEX TARSALIS*/TRAP/WEEK
IN LIGHT TRAPS (5)

Summer Weather in Southern Manitoba, 1981

Hugh M. Fraser
Atmospheric Environment Service
Room 1000 - 266 Graham Avenue
Winnipeg, Manitoba

Abstract

This report describes the weather across southern Manitoba during the summer of 1981. To relate it - in very broad terms - to the subject of concern, the general assumption is made that casual surface water in the spring is conducive to the breeding of *Cx. tarsalis*, and that during the summer rainfall with or followed by warmth further promotes development and activity. In addition, references are made to more specific guidelines suggested by McLintock in 1948 with regard to the reproduction of *Cx. tarsalis* and the transmittal of WEE virus.

Background Weather

According to Agriculture Canada's estimate (Anonymous 1980, 81) soil moisture was above average all across southern Manitoba in the late fall of 1980, and were this followed by an average winter one might anticipate surface pools more numerous than average in the spring. The winter however was surprisingly dry; most areas recorded only half to two-thirds of the long term normal snow amounts. In addition, spring was warm, dry and early, significantly ablating the snow cover. Thus, at the beginning of the season, surface water did not appear to favour the development of *Cx. tarsalis* or at the most would be average in that regard.

To assist in the assessment of weather during the summer, the May through September temperature and precipitation regimes are presented in Figs. 1 and 2 for four Manitoba points. The data was obtained from the "Prairie Provinces Weekly Weather Summary" (Anonymous, 1981) published by the Atmospheric Environment Service of Environment Canada.

May

The dryness of April continued well into May; storm centres carried by west northwest winds high aloft passed for the most part through northern Manitoba. During the last week of the month, the storm track shifted southward and it was a wet week in all but the southwest. Morden reported an exceptional rainfall of over 80mm. Temperatures during the wet period were two to three degrees shy of the 16°C suggested by McLintock as the threshold favourable to *Cx. tarsalis* development. Brandon recorded only a quarter of the long term normal rainfall, and neither warmth nor moisture suggested a mosquito threat in that area.

June

June was typical for the Eastern Prairies as a series of disturbances from the Pacific brought variable weather. Precipitation amounts were near average and in some areas, for example Dauphin, were a little above. As the figures indicate, temperatures were a little less than normal and only consistently above the 16°C threshold in the last week.

July

The first half of July was dry but warm across southern Manitoba. One might speculate that the heat was favourable for mosquito development following the rain of late June. During this period there were no significant storms and winds favoured south or southwest. During the week of the 14th, a low pressure area moved through from the west bringing some rains particularly around Dauphin where 30 mm was reported. Following this disturbance, winds were more northerly and the weather was cooler than average; but because the average temperature is very high at this time of year, the temperatures were still above 16°C.

August

August could be fairly described as wet as a series of low pressure centres swept northeastward from the western U.S.A. across Manitoba. The most vigorous was a storm on August 5 and 6 which brought 30 to 90 mm of rain. After a brief respite, the last half of the month saw more of these disturbances and the southwest part of the province was drenched with up to 100 mm more. Temperatures were variable but generally near average; that average does not fall below 20°C until mid-month at Dauphin and near month-end elsewhere. The weather was probably a factor in extending the mosquito season; although numbers of Cx. tarsalis reported were never high, the peak in population was somewhat less sharp than usual and the decrease was slow in mid-and late August.

September

The storm track from the western U.S.A. to Manitoba shifted southward very early in September but the last of this storm series caught the East of the province with up to 80 mm of rain on the 5th and 6th. There followed two weeks of dry weather with temperatures near or below the normal which falls below 15°C by mid September. Effectively this was the end of the mosquito season.

Discussion

As a general impression, the weather through July did not suggest a large population of Cx. tarsalis. In August as mentioned, there was a broad but low peak in Cx. tarsalis numbers, aided by the rains in the early part of the month; but in perspective the numbers were well below the average peak recorded over the five years since 1977.

McLintock suggests that two weather conditions must prevail before vector mosquitoes are abundant enough to effectively transmit WEE virus in epizootic or epidemic proportions in southern Manitoba: i.e. the average of weekly mean temperatures must be above the normal during the last two weeks of June; and the average of weekly mean temperatures during July and August must be at least 2°C above the normal. These conditions were not met in 1981 but there was a serious WEE threat. The conclusion is that McLintock's guidelines may be pertinent to the development of *Cx. tarsalis*, but that the presence or absence of WEE virus may be independent of the factors that influence mosquito abundance.

References Cited

Agriculture Canada, 1980, 81. Soil Moisture Evaluation Project (SMEP). Agriculture Canada Research Branch, Land Resource Research Institute Ottawa, Ontario.

Atmospheric Environment Service, 1981. Prairie Provinces Weekly Summary. Atmospheric Environment Service, 266 Graham Avenue, Winnipeg, Manitoba. May-September.

McLintock, J. 1948. Report of the Virus Laboratory for 1947. Manitoba Department of Health and Public Welfare. Provincial Laboratories. pp 161-8.

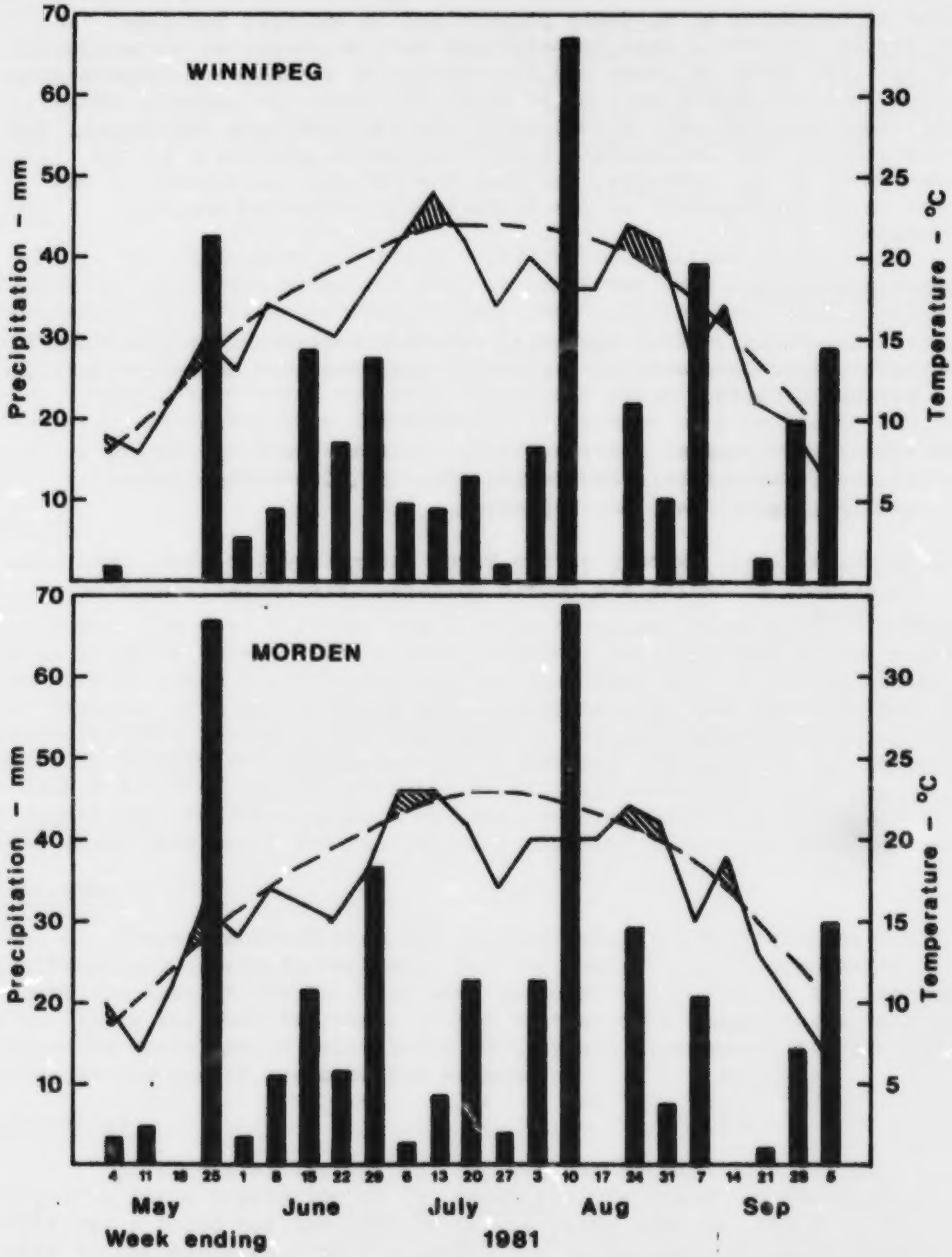


FIG. 1 Summer Weather, Winnipeg and Morden
Temperature: 1981 ____ Normal _ _ Precipitation: 1981 ■

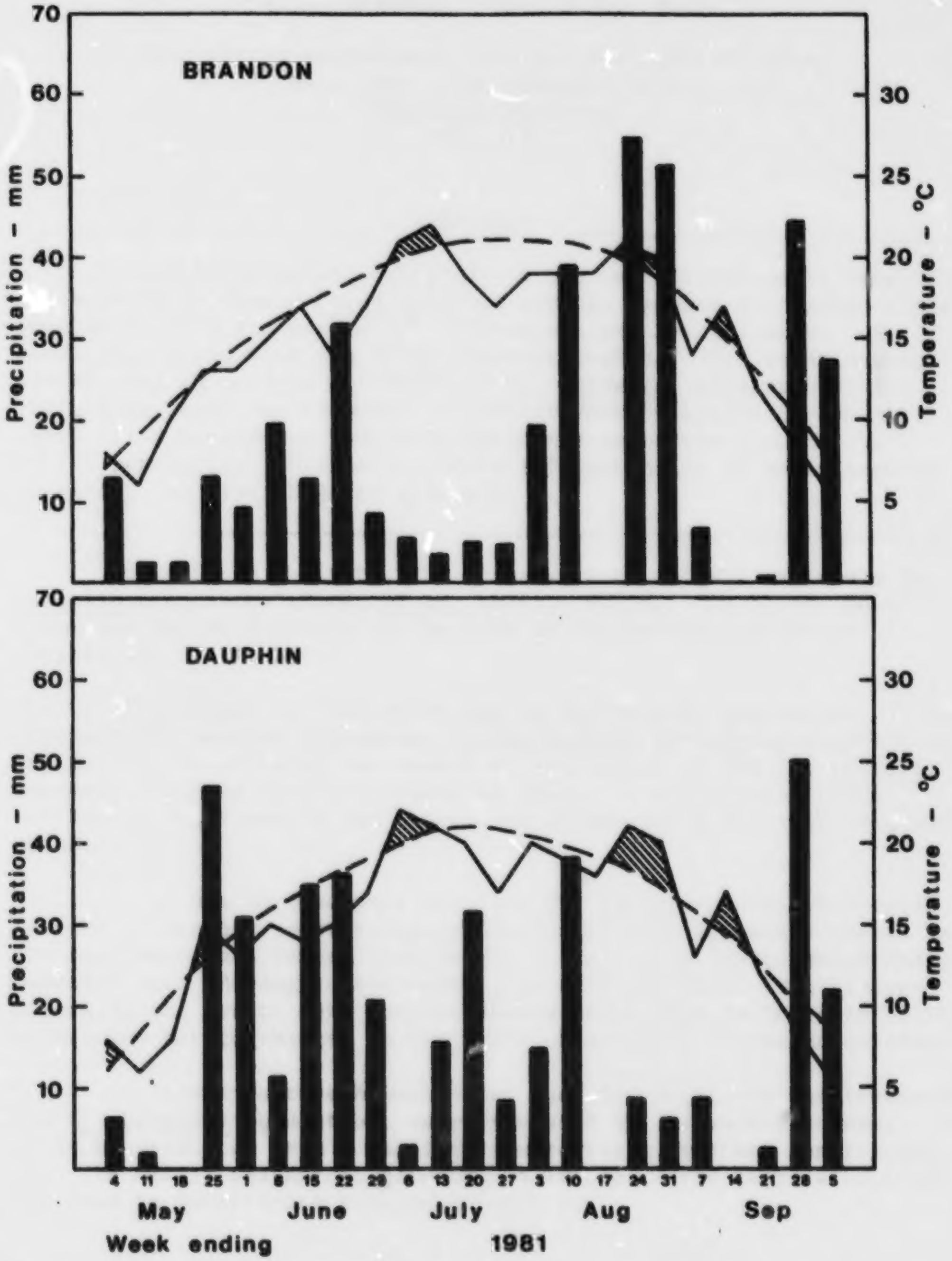


FIG. 2 Summer Weather, Brandon and Dauphin
Temperature: 1981 ____ Normal _ _ Precipitation: 1981 ■

Forecasts of Culex tarsalis Populations in Winnipeg, Manitoba

R. L. Raddatz, B.Sc., B.Ed., M.Sc.

Atmospheric Environment Service, Environment Canada
Room 1000 - 266 Graham Avenue
Winnipeg, Manitoba
R3C 3V4

Abstract

A functional relationship (R = 0.91) was determined between the mean daily female Culex tarsalis counts, assumed to represent the population, and the antecedent temperature and precipitation conditions along with the normal weather regime. This relationship can be used to produce forecasts of Cx. tarsalis populations with sufficient lead-time (8 weeks) to adjust Western Equine Encephalitis (WEE) virus monitoring levels to match the predicted size of the vector population. Ultimately, this information is of assistance in assessing the risk of a WEE epidemic.

In Southern Manitoba, the primary vector of WEE is female Cx. tarsalis mosquitoes (Wood, et al, 1979). Weather may be the most important factor determining the size of the vector population (Fraser and Brust, 1976).

The intent of this study was to investigate the vector population's weather dependency in the hope of developing a method of estimating, in advance, the magnitude and trends of the Cx. tarsalis population. This forecast should be useful in determining virus monitoring requirements and ultimately in assessing the risk of a WEE epidemic.

It has been assumed that the Cx. tarsalis population could be more accurately represented by the longer-term or weekly trends in the capture-counts rather than by the actual daily counts which change markedly from one day to the next. The daily or short-term fluctuations in the counts were not attributed to changes in the size of the population but to changes in the level of activity of the mosquitoes.

It was further hypothesized that the magnitude and trends of the Cx. tarsalis population, as represented by the weekly counts, could be predicted from a leading time-series of weather conditions plus the normal weather or climate. Multiple linear regression (MLR) was used to investigate this relationship.

Non-meteorological effects on the size of the Cx. tarsalis population including the bionomics of the mosquitoes and the impact of control measures, have not been investigated.

2. Vector Data Base

The first step in monitoring and subsequently predicting the Cx. tarsalis population is to obtain a representative sample.

The City of Winnipeg's mosquito surveillence network, consisting of 19 New Jersey style light traps located in and around Winnipeg, provides a data base of Cx. tarsalis capture-counts extending back through the 1977 summer season. Mosquitoes are removed from the traps daily between 8:00 a.m. and noon C.D.T.

3. Vector Populations

Since the daily capture-counts tend to fluctuate significantly from day-to-day, it seems reasonable to treat these daily variations as "noise" which masks the seasonal population curve, that is, the sample time-series representing the population. Therefore, this "noise" was filtered from the sample by averaging the daily counts over one week periods to produce "mean daily counts". One week periods were chosen because weather data is readily available for comparable time periods. Time-averaging resulted in a stepped time-series. That is,

$$\bar{N}(JD) = \left[\sum_{JD}^{JD+7} N(JD) \right] / 7 \text{ days} \qquad (1)$$

where $N(JD)$ = daily Cx. tarsalis capture-count time-series
$\bar{N}(JD)$ = mean daily count time-series representing the population

$JD = 105, 106, \ldots, 243$, the Julian Day from mid-April to the end of August.

It was hypothesized that:

$\bar{N}(JD)$ = f(a leading weather time-series, the climatological time-series)

where f represents "a function of."

The 1977 through 1981 mean daily counts (solid lines) are plotted in Figures 1, 3, 5, 7, and 9. Both 1981 and 1977 were WEE epidemic years even though the time-series of Cx. tarsalis mean counts are considerably different. The counts in 1978 exceeded the 1981 values and were only slightly less than the numbers recorded in 1977, nevertheless, a WEE epidemic did not occur. Relatively few mosquitoes were recorded during the 1979 and 1980 seasons.

4. Predictive Time-Series

For the predictive time-series, weather and climatological parameters were chosen from data recorded at Winnipeg's International Airport and condensed in Environment Canada's Weekly Weather Summary. The parameters included in this bulletin are:

(i) Precipitation (mm) - the week's actual precipitation (P), the week's normal precipitation (PN), the accumulated precipitation since April 1st (PA), and the precipitation normally accumulated from April 1st to date (PNA).

(ii) Temperature (°C) - the week's extreme daily high (TH), the extreme daily low (TL), the average daily mean (T), the normal daily mean (TN) and the difference between daily mean and the normal daily mean temperature accumulated since April 1st then averaged over the number of weeks since that date (ST).

Plots of the 1977 through 1981 mean daily temperatures and total precipitation are indicated in Figures 2, 4, 6, 8 and 10. Normal (1941-1970) values for these parameters are included for comparison.

5. Statistical Estimate of Mean Daily Counts

Multiple linear regression was used to determine a functional relationship between the mean daily Cx. tarsalis time-series, and the antecedent weekly weather parameters plus climatology. The variables were considered separately and in various combinations and several lead-times were investigated. The independent variable most highly correlated with the mean Cx. tarsalis counts was identified and the equation for the line of "best-fit" was calculated. The next variable included in the regression analysis was the one, in combination with the first selected variable, giving a larger multiple correlation coefficient (R) than any other variable paired with the first. In this manner, the technique added variables until the last variable considered for addition to the equation did not contribute significantly to the equation's ability to make predictions.

The statistical estimate of $\bar{N}(JD)$, that is, $\bar{N}(JD)_E$ with the highest multiple correlation coefficient ($R = 0.91$) was obtained using a lead-time of 8 weeks (56 days) for the antecedent weather parameters and the normal mean daily temperature concurrent with $N(JD)$. The equation for the line of best-fit is written below in the predictive sense.

$$\bar{N}\,(JD + 56)_E = -1.381 + 0.006\,(TN(JD + 56) - C)^4 + 0.145 \left\{ \left[(P(JD) + 2.5) - PN(JD) \right] \left[(T(JD) + 1.0) - TN\,(JD) \right] \right\} \quad (2)$$

where

$$C = 13 + A + B$$

The variable A is assigned the appropriate value from Table 1 based on antecedent (8 week lead-time) weather.

TABLE 1

Values for A

Precip. / Temp.	Very Dry $R(JD) < 25\%$	Dry $25\% \leq R(JD) < 50\%$	Moderately Wet $50\% \leq R(JD) < 100\%$	Normal-Wet $R(JD) \geq 100\%$
Cool $ST(JD) < 0$	+2	+1	+1	+1
Normal-Warm $ST(JD) \geq 0$	+2	0	-1	-3

where

$$R(JD) = 100 \times \frac{PA(JD)}{PNA(JD)}$$

$$ST(JD) = \left\{ \sum_{\text{April } 1}^{JD} \left[(T(JD) - TN(JD)) \right] \right\} \Bigg/ \frac{(JD - \text{April } 1)}{7}$$

$B = 0$ except when $P(JD-2) \geq 2.5$ mm and $ST(JD-2) \geq 0$, then B is assigned a value of -3 subject to the following conditions:

(1) when A is assigned a value greater than zero, the value assigned to B is adjusted downward by an equal amount to compensate;

(ii) when B is assigned a value other than zero, the value of B is increased by 1 each subsequent week until B is again zero;
(iii) when A is assigned a negative value, B is reset to zero;
(iv) when TN(JD + 56) reaches a maximum, B is reset to zero.

In equation (2), the results of the differences within the brackets are restricted to values greater than or equal to zero. The precipitation terms are in millimeters and the temperature terms are in degrees Celsius.

This forecast equation can be used to predict Winnipeg's mean daily female *Cx. tarsalis* counts for weeks ending 56 days in the future. The prediction is based on the mean daily temperatures and the precipitation amounts which have occurred up to the current Julian day, JD, plus the normal mean daily temperature for the week ending on the Julian day, JD + 56. All weather terms are known at the time the prediction is made. No weather forecasts, with their inherent uncertainty, are required.

The first term following the constant on the right-hand side of equation (2) accounted for 73% of the total variation in the mean daily counts, that is, the coefficient of determination, R^2, equalled 0.73. The addition of the last term increased R^2 to 0.83. It follows that, the coefficient of alienation, (i.e., $1-R^2$) equalled 0.17. This indicates that 17% of the total variation in $\overline{N}(JD)$ was not accounted for by equation (2). This unexplained variance was attributed to non-meteorological variables and/or to meteorological variables not considered.

The forecast mean daily *Cx. tarsalis* counts (dashed lines) are indicated in Figures 1, 3, 5, 7 and 9. Comparisons with the actual mean daily counts indicates considerable agreement.

6. Discussion and Conclusions

A relationship (R = 0.91) was determined between Winnipeg's mean daily female *Cx. tarsalis* counts and the antecedent temperature and precipitation conditions (8 weeks lead-time) along with the normal weather regime. It has been hypothesized that the mean daily counts represent the *Cx. tarsalis* population. Therefore, this relationship can be used to forecast Winnipeg's *Cx. tarsalis* population with sufficient lead-time to adjust WEE virus monitoring levels to match the expected size of the vector population. Ultimately this information is of assistance in assessing the risk of a WEE epidemic.

The magnitude of Winnipeg's *Cx. tarsalis* population peak, which normally occurs in late July, depends on the precipitation and temperature regimes of the latter part of May. A large July peak (i.e., a mean daily count greater than about 50) occurred when late May was moderately wet to wet with normal to above normal temperatures. Indications are that conditions must be both relatively wet and warm with wet but cold, or warm but dry, conditions producing significantly smaller peaks.

The effect of the weather on the magnitude and trends of *Cx. tarsalis* mean daily counts has been investigated from a statistical rather than an entomological point of view. Therefore, it is not possible to specify why the *Cx. tarsalis* population lags 8 weeks behind the weather. One can speculate, however, that some "snowball effect" is involved. That is, with Winnipeg's short spring and summer seasons, if the *Cx. tarsalis* population doesn't get an early start, the season isn't long enough to build a small spring population into a large summer population.

The results presented above deal with the dependent or developmental sample, that is, the 1977 through 1981 mosquito seasons in Winnipeg. The forecast equation was employed in 1982 with that mosquito season serving as an independent data set or "test". Results for 1982 are included in Appendix I.

Acknowledgements

I wish to thank Dr. R. Ellis, City of Winnipeg, Entomologist for providing the daily *Culex tarsalis* capture-count data. Thanks are also due Steve MacPherson of Enviroment Canada, Winnipeg, for his assistance in the analysis of the data.

References Cited

1. FRASER, H.M. and R. A. BRUST, 1976. Weather Conditions affecting Mosquito Populations in Southern Manitoba during 1975. *Canadian Journal of Public Health*, *Supplement one. 67*: 40-46.

2. WOOD, D.M. *et al*, 1979. The Insects and Arachnids of Canada, Part 6, The Mosquitoes of Canada. Canadian Government Publishing Centre, Hull. 390 pp.

APPENDIX I

Culex tarsalis Forecasts for Winnipeg - 1982

1. Verification

The predicted mean daily *Cx. tarsalis* counts for 1982 along with the 68%, 95% and 99.7% confidence intervals are shown in Figure 11. The confidence intervals were derived from the standard error of estimate which has a value of 17. The actual mean daily *Cx. tarsalis* counts are also plotted. Figure 12 is a summary of 1982's mean temperature and precipitation values.

A comparison of the forecast and actual mean daily counts indicates some early season successes, however, the forecasts departed markedly from the actual counts towards the end of July. The actual counts, nevertheless, remained within or close to the 99.7% confidence interval. This implies that the deviations were due to the residual variance and to uncertainty in estimation rather than to model biases. Verification of 1982's forecasts indicates that the accuracy of the model is such that the mean daily *Cx. tarsalis* counts can only be predicted to within relatively broad intervals, with the intervals shrinking as the confidence in the forecast is reduced.

2. Interpretation

A WEE epidemic did not occur in 1982 even though *Cx. tarsalis* were in relative abundance in the latter part of July and early August. *Cx. tarsalis* were few in number up to that point. A comparison with the developmental sample showed 1978 to be the closest analogue. That year also had a relatively large peak in mean daily *Cx. tarsalis* counts in the latter half of July with relatively few mosquitoes up to that time and a WEE epidemic did not occur. An examination of the epidemic years, 1977 and 1981, suggested that a *Cx. tarsalis* peak greater than about 50 was sufficient to transmit the WEE virus in epidemic proportions. Comparisons between these years and 1978 and 1982 revealed that the "significant" rise in *Cx. tarsalis* mean counts occurred earlier in the epidemic than in the non-epidemic years (i.e., late June or early July versus late July). The years 1979 and 1980 produced relatively few *Cx. tarsalis* and no epidemics. Thus, it appears that two conditions must be met before *Cx. tarsalis* transmit the WEE virus in epidemic proportions. These are, (1) a peak in the mean counts greater than about 50, and (2) an early increase in the counts to this significant number.

If the 1982 forecasts are again considered from the 'a priori' viewpoint, it can be seen that neither of the conditions delineated above would be met by actual counts which fell within the 68% confidence interval. However, both conditions would be met with actual counts falling within the higher confidence intervals. That is, the forecast odds against the occurrence of a WEE epidemic in 1982 were only 2:1.

The sample is small (6 years) and other interpretation of the apparent patterns in the data are possible. However, it appears that to within specified confidence limits, predictions are useful in giving an early indication that *Cx. tarsalis* will or will not be in sufficient numbers, at an early enough date, to transmit the WEE virus in epidemic proportions.

With a firm base in quantified experience now established, it is anticipated that more accurate predictions and interpretations can be made in the future.

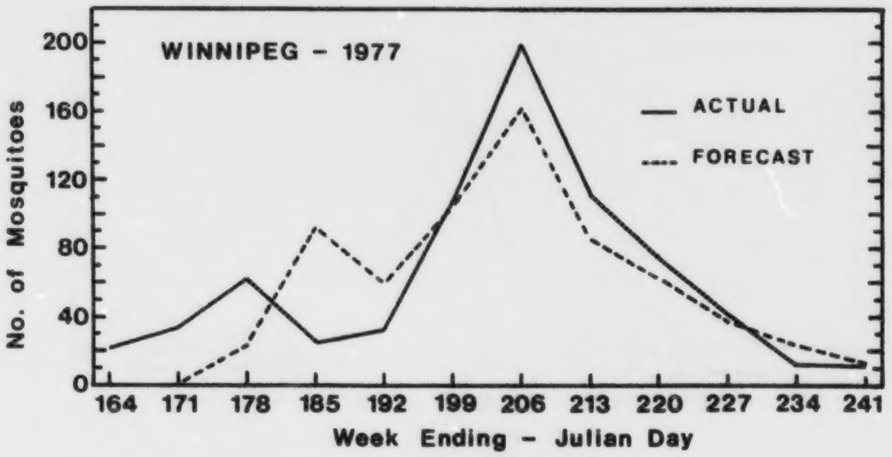


FIG. 1 Culex tarsalis (Female) Count

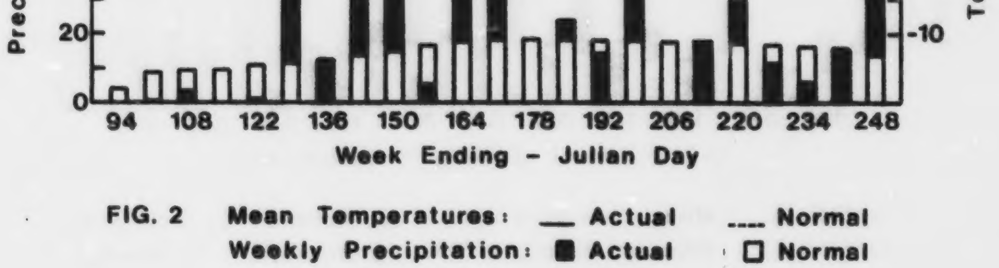


FIG. 2 Mean Temperatures: — Actual ---- Normal
Weekly Precipitation: ■ Actual □ Normal

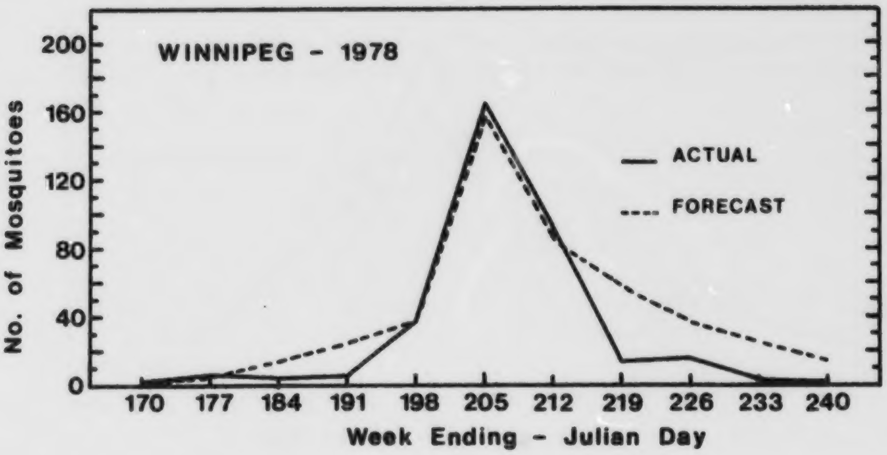


FIG. 3 *Culex tarsalis* (Female) Count

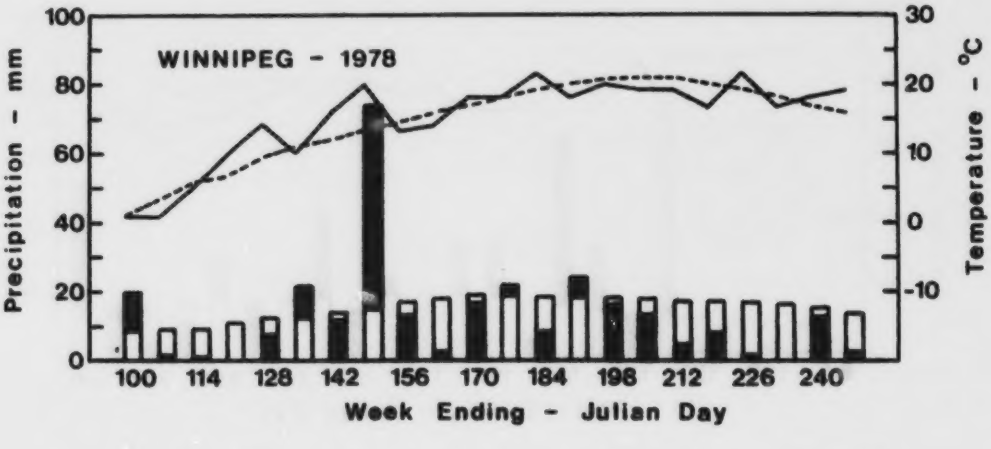


FIG. 4 Mean Temperatures: ___ Actual ---- Normal
Weekly Precipitation: ■ Actual □ Normal

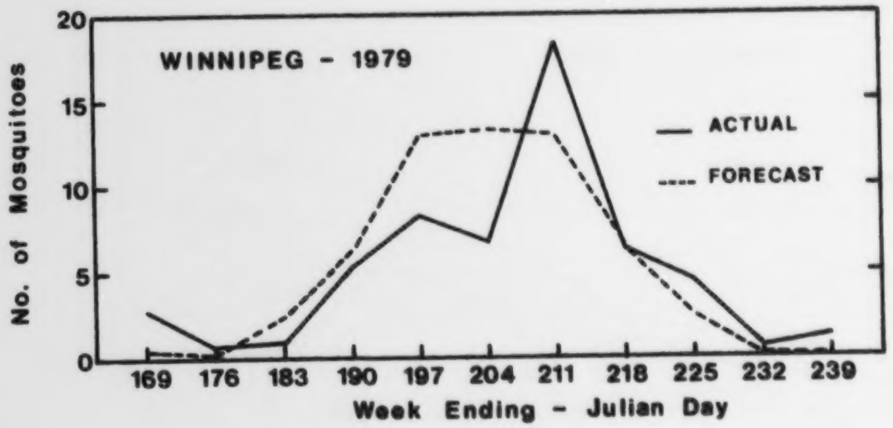


FIG. 5 *Culex Tarsalis* (Female) Count

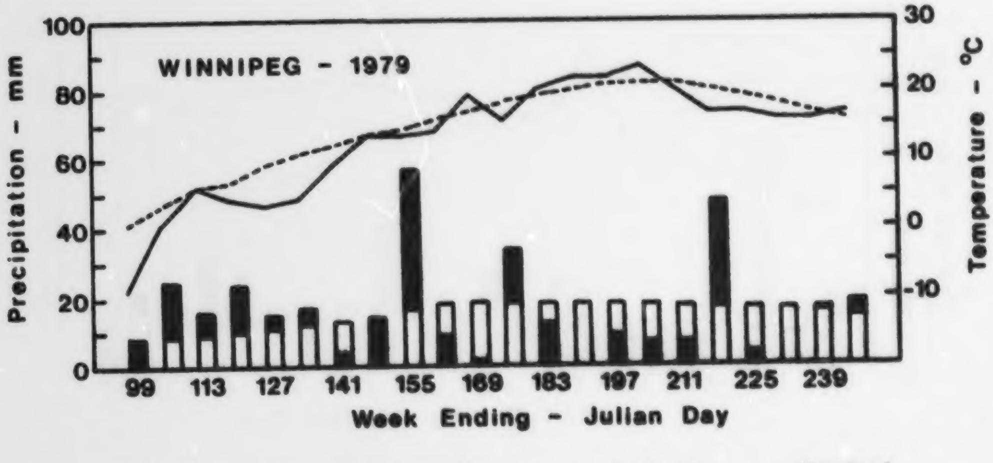


FIG. 6 Mean Temperatures: ___ Actual ---- Normal
Weekly Precipitation: ■ Actual □ Normal

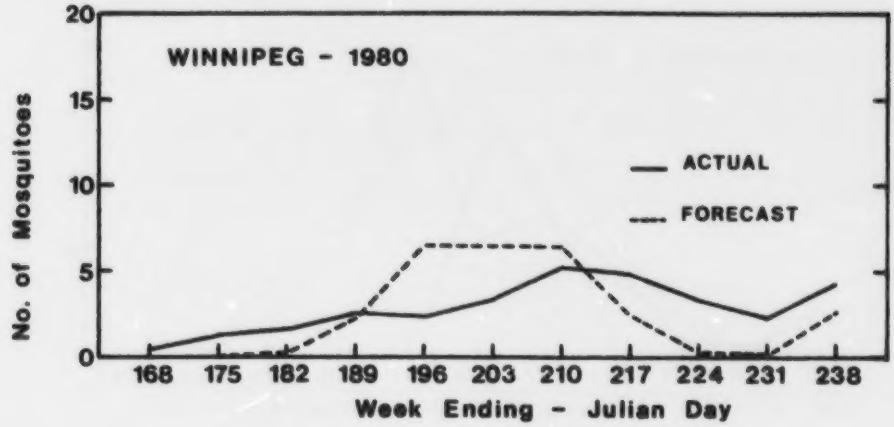


FIG. 7 *Culex tarsalis* (Female) Count

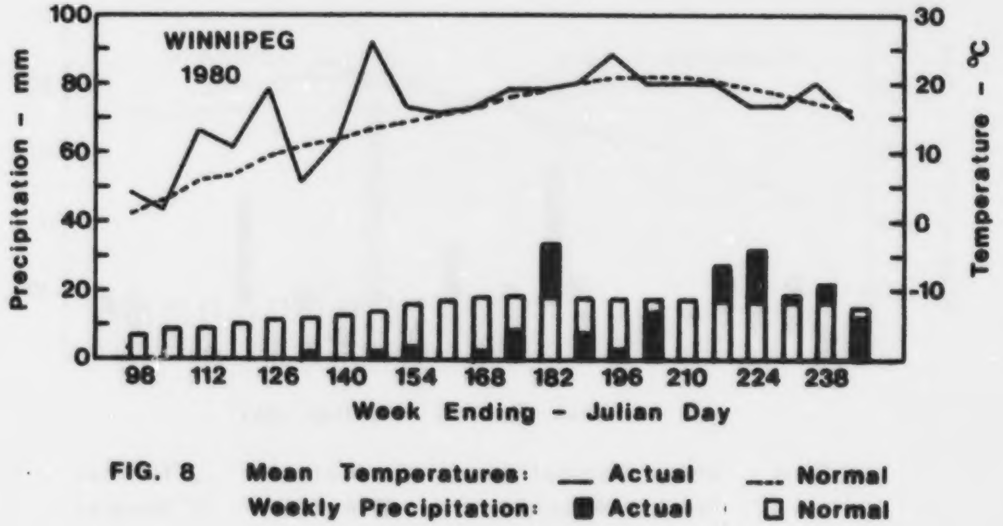


FIG. 8 Mean Temperatures: ___ Actual ---- Normal
Weekly Precipitation: ■ Actual □ Normal

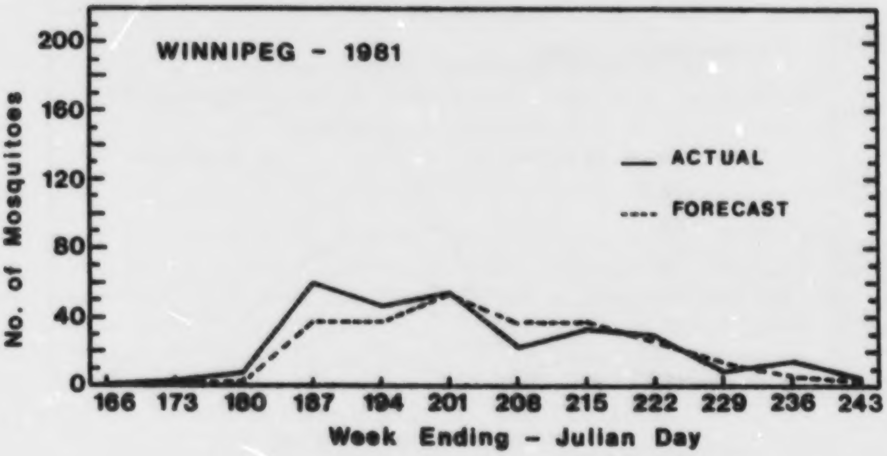


FIG. 9 Culex †arsalis (Female) Count

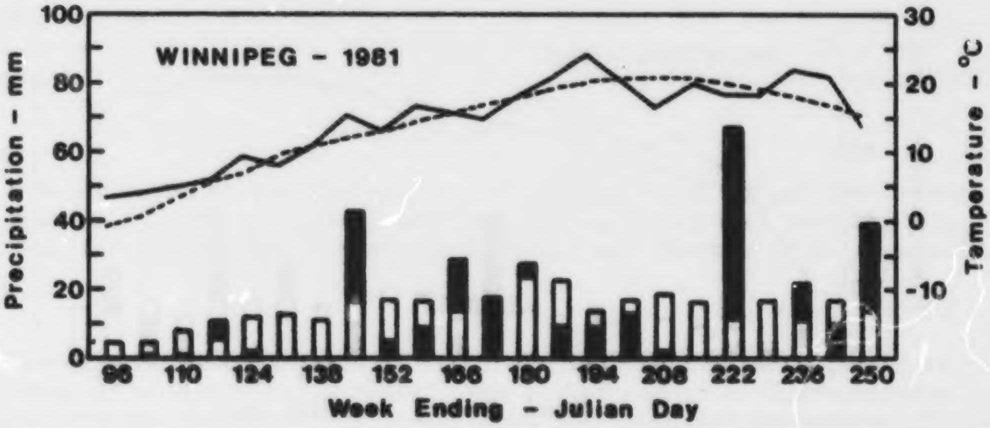


FIG. 10 Mean Temperatures : ___ Actual --- Normal
Weekly Precipitation : ■ Actual □ Normal

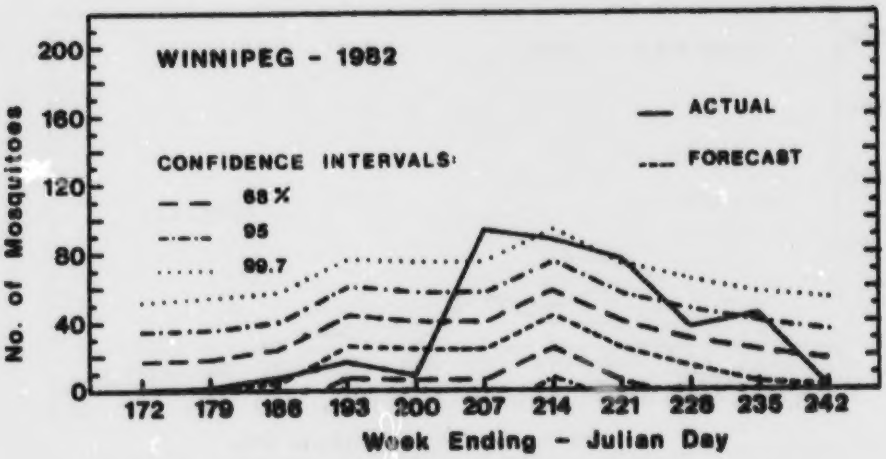


FIG. 11 *Culex Tarsalis* (Female) Count

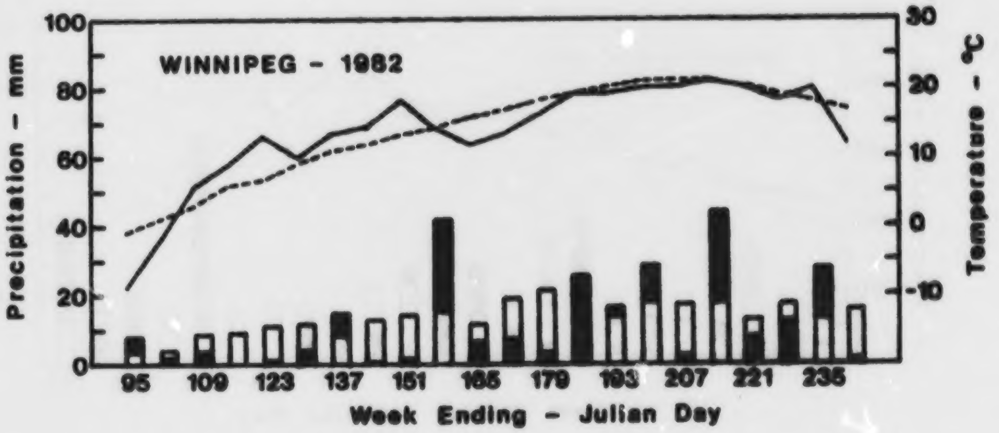


FIG. 12 Mean Temperatures: ___ Actual ---- Normal
Weekly Precipitation: ■ Actual □ Normal

Arbovirus Isolations from Mosquitoes in Manitoba: Value in Decision-Making

L. Sekla M.B.B.Ch., Ph.D.
W. Stackiw, B.Sc.

Manitoba Health Services Commission
Cadham Provincial Laboratory 750 William Avenue
Winnipeg, Manitoba R3C 3Y1

Abstract

A total of 113 Western Equine Encephalitis (WEE), 2 California Encephalitis (CE), 4 Cache Valley (CV) and 2 Hart Park Flanders (HPF) viruses were isolated from field-collected mosquitoes during the years 1975 - 1981. A variety of female mosquito species were found to be infected with arboviruses; Culex tarsalis is the main vector associated with epidemics of WEE in Manitoba. In 1975, 1977 and 1981 the infection was widespread in the Southern part of the Province.

Arbovirus isolations can be of value in predicting epidemics of WEE. During the years 1975 - 1981 marked differences were noticed between epidemic and non-epidemic years pointing out to a clear association between the detection of infected Culex tarsalis and epidemics of WEE in Manitoba. Arboviruses were isolated from field mosquitoes collected post-aerial spraying with an adulticide.

Western Equine Encephalitis (WEE) was isolated for the first time in Manitoba in 1942 (Norris 1946); this and subsequent isolations were reviewed briefly (Sekla and Stackiw 1976); (Sekla, Stackiw and Brust 1980).

MATERIALS AND METHODS

A description of the collection and submission of mosquitoes is presented by Brust 1982 (In press) and that of the procedures used for arbovirus isolations from mosquitoes by Sekla and Stackiw 1982 (In press).

RESULTS

The data obtained in each of the years 1975 to 1981 with regard to the number of mosquito pools tested, the number and type of arboviruses isolated and the percentage of pools found positive, are summarized (Table 1).

A total of 4,153 pools of mosquitoes were tested. A total of 121 arboviruses were isolated; of these 113 were WEE, 2 were California Encephalitis (CE), 4 were Cache Valley (CV) and 2 Hart Park Flanders (HPF). The 2 CE viruses were identified by the Colorado Vector Borne Centre and the National Arbovirus Reference Center as a Snowshoe hare and a JC sub-types. No viruses were isolated from mosquitoes in 1976 and 1980; only 1 WEE was isolated in 1978, while in 1979, 4 WEE, 1 HFP and 2 CE viruses were isolated. The number of arbovirus isolations during the above non-epidemic years (8 in all 4 years) contrasts markedly with the 113 isolated in the 3 epidemic years, 1975, 1977 and 1981.

A variety of species of mosquitoes were naturally infected with arboviruses (Table 2). The table shows the range of adults per pool of mosquitoes tested, the type of arbovirus isolation, the year of isolation, and the minimum infective ratio (MIR). The latter can be easily converted into MIR per thousand mosquitoes by multiplying the figure quoted in Table 2 by 1,000.

The time of collection of the infected mosquitoes,indicating that all the isolations were made during the months of July and August,is shown (Table 3).

The WEE virus isolations from mosquitoes during the years 1976 and 1981, the total numbers of pools and mosquitoes tested, the percentage of *Culex tarsalis* pools and mosquitoes tested, as well as the percentage of *Culex tarsalis* found positive are shown (Table 4). Clearly *Culex tarsalis* is the vector associated with WEE epidemics in Manitoba.

The localities from which arbovirus-infected mosquitoes were collected for each of the years 1975 - 1981 are shown (Table 5). Table 5 illustrates that in epidemic years the infection is widespread.

Following aerial spraying, mosquitoes were collected and tested for viruses in 1975, 1977 and 1981. The number of pools tested, the number of Culex tarsalis pools and mosquitoes collected, the date of collection, and the number and type of arboviruses isolated for each of the 3 years under study are shown (Table 6). A total of 231 pools of mosquitoes were tested and only 2 arboviruses were isolated from mosquitoes collected post-spraying. In 1981, 32.5% of the mosquitoes collected post-spraying were *Culex tarsalis*. A WEE virus was isolated from *Culex tarsalis* caught in Portage la Prairie on August 12, 1981 and a Cache Valley virus isolated-from *Culiseta inornata* caught in Portage on August 17, 1982. The infected *Culex tarsalis* pool may have been a mixture of pre- and post-spraying populations.

Analysis of Results

1. Table 1 shows a sharp difference in the percentage of positive mosquito pools between epidemic and non-epidemic years. In non-epidemic years less than 2% of the mosquito pools tested carried arboviruses, compared to epidemic years where it was 4.8 to 7.0%.

2. Table 2 shows that Culex tarsalis is the main vector of WEE (carrying 45% of the WEE viruses isolated), followed by Culiseta inornata and Aedes vexans. WEE virus was isolated from Aedes vexans (Meigan) and Culiseta inornata (Williston) for the first time in Manitoba in 1977 (Sekla et al 1980); since then the virus has been found in these same species in 1978, 1979 and 1981. By contrast, the WEE virus was isolated from Cq pertubans (Walker) and Aedes pionips (Dyar) in 1977 only, being first reports from these species. Virus isolation from Anopheles earlei (Vargas) was also reported for the first time in 1977; the WEE virus was isolated in 1979 and again in 1981 when a pool of 2 mosquitoes were found to be positive.

3. Culex tarsalis was found to be carrying the HPF viruses in 1979 and again in 1981. Culiseta inornata appears to be the vector for the CV viruses isolated in 1977 and 1981. Culiseta inornata also carried a CE virus in 1979, while Aedes spp were found to be infected with this virus in 1979.

4. Table 2 shows that the WEE virus has been isolated from pools containing as few as 1 - 2 mosquitoes.

5. The MIR assumes that at least 1 mosquito is infected in each positive pool compared to the total number of adults of that species that were tested. It is interesting to note that in 1977 Anopheles earlei and Cq pertubans had MIR higher than that of Cx tarsalis (respectively 1:87, 1:202 and 1:347). In 1981, the MIR for Anopheles earlei was 1:59, but only 1 of the 30 pools tested harboured the WEE virus. It would be useful to determine the exact role of these mosquitoes in the transmission of WEE.

6. McLintock (McLintock and Iversen 1975) has suggested that Aedes spp may be involved in the amplification of WEE virus early in the spring. However, no arboviruses were isolated from any species of mosquitoes prior to the first week of July in any of the years 1975 - 1981. This is illustrated in Table 3 in which arrows indicate the beginning and the end of the surveillance period for each of the years under study. It is worth mentioning that no virus was isolated from mosquitoes caught after August 24, in any of these years. The infective season in Manitoba is a brief one. Virus isolations from mosquitoes could be restricted to July and August and extended to June or September only when weather conditions favourable to the amplification of the mosquito vectors develop during these months.

7. Table 4 shows that the percentage of Culex tarsalis found to be infected with WEE in epidemic years (10.2 in 1977 and 8.5 in 1981) contrasts sharply with the total lack of isolations from Culex tarsalis in the non-epidemic years 1976, 1978, 1979 and 1980. It

is worth stressing that the WEE virus was isolated once in 1978 and 4 times in 1979 (2 non-epidemic years), but in both years the WEE isolations were made from species of mosquitoes other than Culex tarsalis. Clearly, in Manitoba epidemics of WEE are associated with the isolation of WEE virus from Culex tarsalis. In epidemic years, at least 8% of the Culex tarsalis pools tested carried the virus.

8. Table 5 details the locations from which mosquitoes were collected in each of the years 1975 - 1981, also showing the types of arboviruses isolated wherever relevant. The fact that virus isolation requires the processing of either live mosquitoes or frozen in ampoules on dry-ice has restricted the area covered by the surveillance to the Southern part of the Province. The number of locations monitored varied from year to year in proportion to the budget allocated to the surveillance. More than 1 type of arbovirus has been isolated from the same location, as for example WEE and HPF, from the Charleswood traps. Both CE viruses were isolated from Selkirk and Oak Hammock, 2 neighbouring locations. Table 5 clearly shows that during epidemic years WEE infections are widespread.

Predictive Value of Culex tarsalis Indices

A prospective use of the Culex tarsalis data to predict and monitor WEE epidemics could be of great assistance to the surveillance program. To determine the most pertinent Culex tarsalis indices, the information gathered from 1976 - 1981 was analysed separately for each week of July and August for each of the following 3 parameters: 1) Culex tarsalis expressed as percentage of the pools of mosquitoes tested for virus isolation. 2) Culex tarsalis expressed as percentage of the actual number of mosquitoes tested. 3) Percent Culex tarsalis found infected with WEE virus. A critical level was arbitrarily selected for each of these 3 parameters: 25%, 25% and 2% respectively. Figure 1 shows that for the first 2 parameters the critical level of 25% was clearly exceeded in the last 3 weeks of July 1977 and in all 4 weeks of July 1981 (both WEE epidemic years), as well as in the last week of July 1979, a non-epidemic year. However, for the third parameter, the critical level of 2% was exceeded in 1977 and 1981, the 2 epidemic years. Therefore, it may be inferred that when these 3 parameters are taken simultaneously then it can be of some predictive value. During the month of July, if at any given week, 25% of the mosquito population consists of Culex tarasalis and if 2% of these Culex tarsalis are infected, than an epidemic of WEE is either impending or occurring. The above criteria appear to be valid for the month of August, as well as July as may be seen in Figure 2. All 3 critical levels were exceeded only in the first 2 weeks of August 1977 and 1981. It is worth mentioning that the percentage of infected Culex tarsalis was well above the 2% critical level in July and August 1977 and 1981; in fact the lowest level of infectivity during an epidemic was reached during the first week of August 1977 when only 4% of the Culex tarsalis were infected.

Value of Arbovirus Isolations from Mosquitoes in Predicting WEE Epidemics

In setting up a surveillance program it is important to have an adequate number of mosquito trapping locations which could be monitored every year to facilitate data collection and trend analysis. Though in epidemic years the infection is widespread, it may develop more slowly or require more time to be detected in some locations.

During the years 1975 - 1981, arbovirus isolations from mosquitoes proved to be useful in predicting epidemics of WEE. Table 6 summarizes the data already presented and clearly shows that:

1. The percentage of mosquito pools infected with arboviruses is clearly higher in epidemic years. In non-epidemic years (1976, 1978, 1979 and 1980) less than 2% of the mosquito pools tested were found to be positive.
2. The percentage of positive Culex tarsalis pools was 8.5% or greater in epidemic years and nil in non-epidemic years.
3. The WEE virus was isolated from mosquito species other than Culex tarsalis in epidemic years as well as in non-epidemic years.

SUMMARY

Arbovirus isolations from mosquitoes can be of value in predicting epidemic of WEE. Culex tarsalis is the main vector of WEE in Manitoba.

References Cited

Brust, R.A., 1982. Population Dynamics of C. tarsalis Coquillet (In press).

McLintock, J., Iversen, J., 1975. Mosquitoes and Human Diseases in Canada. Can. Ent. 107:695-704.

Norris, M., 1946. Recovery of a strain of Western Equine Encephalitis from a Culex restuans. (Theo) (Diptera: Culicidae). Can. J. Res. (E)24:63-70.

Sekla, L., Stackiw, W., 1976. Laboratory Diagnosis of Western Encephalomyelitis. Can. J. Public Health. Suppl. 1 to Vol. 67.

Sekla, L., Stackiw, W. and Brust, R., 1980. Arbovirus Isolation from Mosquitoes in Manitoba. Mosq. News. 40:377-380.

Sekla, L. and Stackiw, W., 1982. Arbovirus Isolations and Serology (In press).

TABLE 1

ARBOVIRUS ISOLATIONS FROM MOSQUITOES

	1975	1976	1977	1978	1979	1980	1981	
POOLS TESTED	72	882	1045	581	450	395	728	= 4153
ARBOVIRUS ISOLATED	5	0	73	1	7	0	35	= 121
% POOLS POSITIVE	7	–	7	0.1	1.6	–	4.8	
WEE	5	–	71	1	4	–	32	= 113
HPF	–	–	–		1	–	1	
CVV	–	–	2	–	–	–	2	
CEV	–	–	–	–	2	–	–	

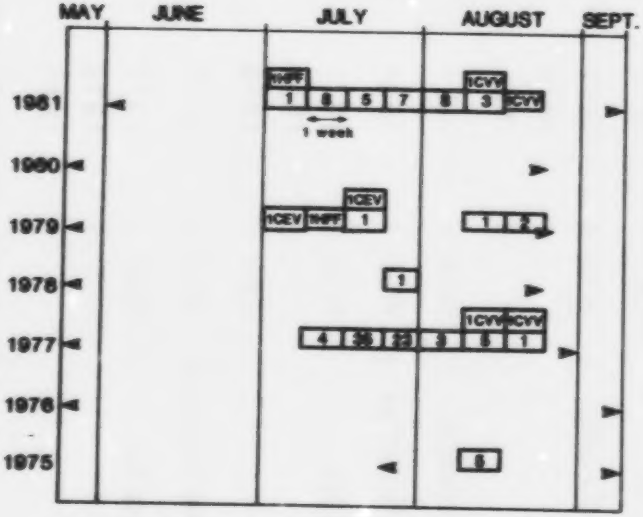
TABLE 2
SPECIES OF MOSQUITOES NATURALLY INFECTED

			RANGE OF ADULTS PER POOL	MINIMUM INFECTIVE RATIO*
Mixed spp.	WEE 1	1976	16-260	
Cx. tarsalis	WEE 33	1977	9-60	1:347
	18	1981	4-60	1:283
	HPF 1	1979	21	1:1347
	1	1981	10	1:13163
Culex spp.	WEE 9	1977	26-60	1:371
Cu. inornata	WEE 8	1977	4-64	1:706
	3	1979	80	1:764
	6	1981	1-41	1:793
	CVV 2	1977	1-2	1:2729
	2	1981	17-50	1:2360
	CEV 1	1979	8	1:1282
Ae. vexans	WEE 8	1977	28-87	1:761
	1	1978	80	1:9208
	3	1981	18-60	1:1282
Ae. pionips	WEE 1	1977	60	1:2773
Ae. sticticus	WEE 1	1981	1	1:217
Ae. spp.	WEE 8	1977	28-87	1:1761
	2	1981	2-68	1:416
	CEV 1	1979	64	1:6784
Cq..pertubans	WEE 1	1977	7	1:202
An. earlei	WEE 3	1977	4-6	1:67
	1	1979	14	1:42
	1	1981	2	1:53

* To standardize MIR multiply by 1000

TABLE 3

TIME OF COLLECTION - No. POSITIVE POOLS



CVV = Cache Valley
CEV = California Encephalitis
HPF = Hart Park Flanders
WEE = Western Equine Encephalitis
Arrow Heads = Beginning and End of Collection

WEE

TABLE 4

Cx tarsalis DATA - MANITOBA 1976-1981

	1976	1977	1978	1979	1980	1981
- Total No. tested						
Pools	822	1046	581	450	395	728
Mosquitoes	9492	36243	24758	11272	1887	14073
- % Cx tarsalis*						
Pools	19.3	30.8	11.1	13.5	22.7	28.9
Mosquitoes	12.8	36.2	11.5	10.3	17	36.2
- % Cx tarsalis ⊕ pools	0	10.2	0	0	0	8.5
- No. of WEE-isolations from						
Cx tarsalis	0	33	0	0	0	18
Other spp.	0	38	1	4	0	14

*Pools: 100% Cx tarsalis females

TABLE 5

COLLECTION LOCATIONS α ARBOVIRUS ISOLATIONS- MOSQUITOES

Location	1975	1976	1977	1978	1979	1980	1981
Birds Hill	X						
Brandon		X	☒	X	X	X	X
Charleswood	☒	X	☒	☒	☒	X	☒
Delta	X						
Glenlea						X	☒
Melita		X					
Morris		X	☒		X		
Oak Hammock			☒	X	☒	X	☒
Portage	X	X	☒	X	☒	X	☒
Selkirk		X	☒	X	☒	X	☒
Stonewall	☒				X		
U. of Manitoba		X	☒	X		X	☒

☐ Western Equine Encephalitis

☐ Hart Park Flanders

☐ California Encephalitis

☐ Cache Valley

TABLE 6

POST-SPRAYING - ARBOVIRUS ISOLATIONS

	1975	1977	1981
Number of pools tested	26	47	160
Cx tarsalis No. /mosquitoes	1/1	12/23	52/426
Date of Collection	27/8-5/9	24/8-31/8	12/8-15/9
Arbovirus isolations	0	0	1 WEE 1 CVV

☐ Cx tarsalis- Portage
Mixture of pre and post-spraying populations

CVV Cu. inornata- Portage

CVV = Cache Valley
WEE = Western Equine Encephalitis

TABLE 7

SUMMARY OF DATA ON ARBOVIRUS ISOLATIONS FROM MOSQUITOES 1975-1981

		1975*	1976	1977*	1978	1979	1980	1981*
I	Percentage of positive mosquito pools	7	0	7	0.17	1.6	0	4.8
II	Percentage of positive Culex tarsalis pools	ND	0	10.2	0	0	0	8.5
III	Number of WEE isolations from:							
	a) Culex tarsalis	ND	0	33	0	0	0	15
	b) Other species of mosquitoes	ND	0	38	1	4	0	14
IV	Number of isolations of other arboviruses	0	0	2 CV	0	1 HPF 2 CE	0	1 HPF 2 CV

WEE = Western Equine Encephalitis Virus

CV = Cache Valley Virus

HPF = Hart Park Flanders Virus

CE = California Encephalitis Virus

* = Epidemic Year

ND = Not Done

Figure 1 : Cx tarsalis in July

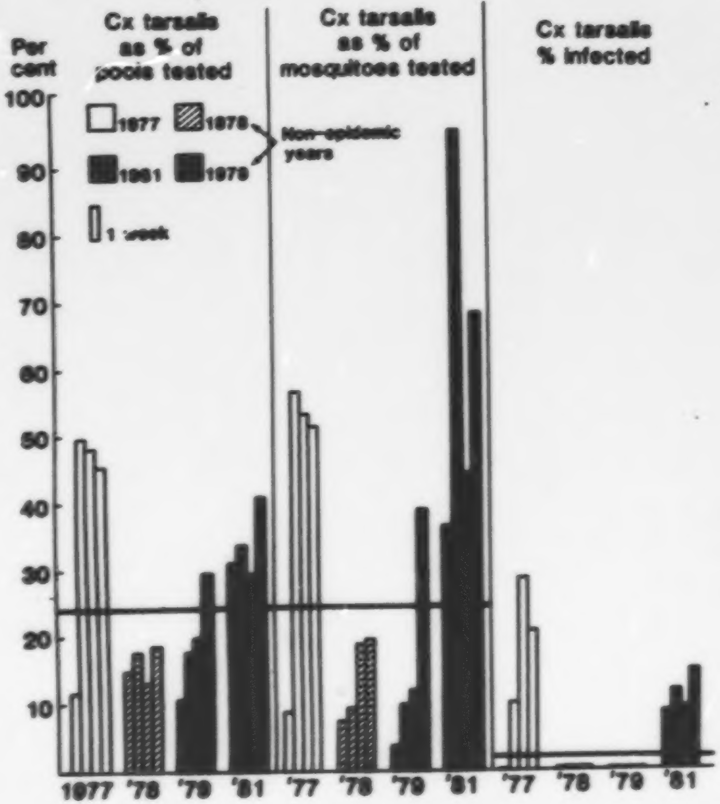
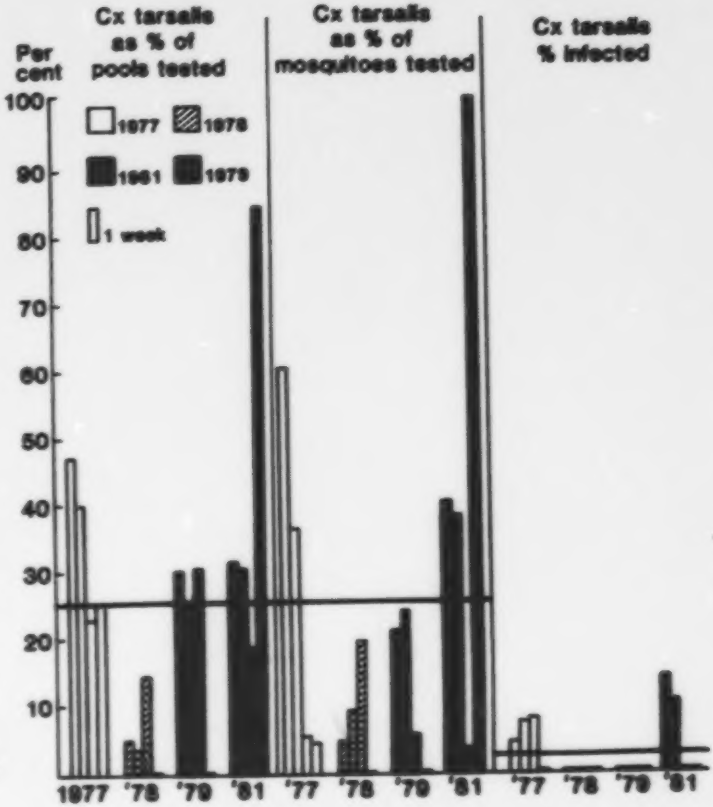


Figure 2 : Cx tarsalis in August



CHAPTER III:

Equine Data

Western Equine Encephalomyelitis in
Manitoba - Equine Cases, 1975-1981

J.L. Neufeld, D.V.M., Diplomate A.C.V.P.
G.P.S. Nayar, D.V.M., M.Sc., Diplomate A.C.V.M.

Veterinary Services Branch
Manitoba Department of Agriculture
545 University Crescent
Winnipeg, Manitoba
R3T 2N2

Abstract

Horses were afflicted with Western Equine Encephalomyelitis (WEE) in 1975, 1977 and 1981. Confirmed cases numbered 139, 53 and 128 respectively. In 1981, there were 27 deaths compared to 6 in 1975 and 4 in 1977. Horse cases appeared approximately 1 week after sentinel flocks seroconverted. Human cases followed horse cases by about 2 weeks in the 3 outbreak years. Southeastern Manitoba and the Red River Valley region appear to be high risk areas, as shown by the infection rates compiled for each of the crop districts. The severity of the 1981 outbreak and its relationship to the vaccination status of the horse population are discussed. The criteria used to confirm positive cases and the possibility of using the horse as a sentinel species for the detection of WEE virus activity in the environment are described.

An extremely severe disease affecting the central nervous system (CNS) of horses caused devastating losses on the prairies of Western Canada during the decade 1930-1940 caused by an arbovirus named Western Equine Encephalomyelitis (WEE) virus. Periodically, the virus continues to cause outbreaks of disease in horses and humans when conditions are conducive to viral transmission (Lillie *et al* 1976).

This report deals with WEE in Manitoba from 1975 to 1981. During 1975, 1977 and 1981, numerous horses were affected, many of which died. The data gathered during these 7 years are presented in a comparative format.

MATERIALS AND METHODS

Submission of Samples

Veterinarians in the Province of Manitoba were encouraged to submit blood samples from suspected cases of WEE to the Provincial Veterinary Laboratory (PVL). They were requested to submit a blood sample taken during the acute phase of the disease from each suspect case. The practitioners were also instructed to submit a second blood sample taken 2-4 weeks after the acute phase. If any of the affected horses died, the PVL encouraged the practitioners to submit brain tissue for histopathological and virological studies.

Serology

The clotted blood samples were centrifuged at 2,000 rpm for 10-15 minutes to separate the serum. The sera, thus separated, were then tested by hemagglutination-inhibition (HI) procedures (Sekla and Stackiw 1976). HI positive sera were tested for complement fixing antibodies (CF) as well. A few selected sera were also tested at the Animal Diseases Research Institute (A.D.R.I.) in Ottawa to detect levels of complement-fixing antibody against both WEE and Eastern Equine Encephalomyelitis (EEE).

Histopathology and Virus Isolation

Veterinary clinicians submitted brain tissue from some dead horses in a fresh state or fixed in 10% formalin. Fixed tissues were processed using standard procedures of dehydration and embedding. Paraffin sections were cut at 6 microns thickness and stained with haematoxylin and eosin prior to histopathological examination.

Fresh tissue was submitted to the A.D.R.I. for immunofluorescence testing for rabies antigen. Virus isolation procedures were conducted on all fresh tissue samples either at the PVL or at the Cadham Provincial Laboratory (CPL), using standard virological procedures previously reported (Lillie et al 1976; Sekla and Stackiw 1976).

Data Evaluation Procedures

Data from the 1975 WEE outbreak in Manitoba (Lillie et al 1976) have been regrouped to compare them with 1977 and 1981.

Census data of horses on farms is available from Statistics Canada, the latest figures being for 1981. The agricultural area of Southern Manitoba is arranged into 12 crop reporting districts. The number of horses in each district is shown (Fig. 1). Using the population figure for each district and the number of corresponding WEE cases, the infection rate pqr 1,000 horses was calculated for each district. The infection rate was then used to compare districts and to identify the district having the highest rate. The fluctuations in horse population over the years is shown (Fig. 2).

For proper interpretation of the data, it is important to know the date of arrival of blood samples from clinical cases of WEE and the number of samples arriving per week. The total number of suspect cases arriving per week and the number of confirmed cases were tabulated. This is useful when comparing WEE incidence in different years and is also useful in determining whether or not an outbreak is escalating or declining in severity.

The age distribution of horses affected, available for 1977 and 1981 only, and the number of vaccinated affected ones are shown (Fig. 3).

Confirmation of WEE Virus Infection

In order to confirm WEE virus infections in a horse the following criteria were used: i.e.

1. Paired sera must show a 4-fold or greater increase in antibody titre, either by CF or by HI test;
2. a single serum sample from a horse under 1 year of age, with no history of vaccination but showing CNS signs, must have an antibody titre of 1:64 or greater when tested, using the HI test;
3. a single serum sample from a horse, regardless of age and immunization status must have an antibody titre of 1:256 or greater;
4. a single serum sample from a horse with a history of vaccination must have an antibody titre of 1:1024 or greater;
5. brain tissue must have lesions of non-suppurative encephalitis as revealed by histopathological examination and must be negative for other etiologic agents such as rabies;
6. if a viral agent is successfully isolated from brain tissue, it must be confirmed by neutralization with WEE antisera, using standard virus neutralization procedures.

Cases not meeting one or more of the above criteria were considered negative.

RESULTS

The number of cases submitted, confirmed and deaths recorded each year are given, (Table 1). The years of highest incidence are 1975, 1977 and 1981. The frequency of case submissions is given, (Fig. 4). In an outbreak year, the cases invariably begin to escalate in late July and to peak in mid-August. Each time the number of cases exceeded 6 per week and increasing numbers of samples were received at the PVL. Once the peak numbers were reached, there was either a rapid (1975) or a more gradual reduction (1977 and 1981).

In non-outbreak years, the number of suspect cases ranged from 19 in 1976 to 5 in 1978. Confirmed cases ranged from 0 - 10 (Table 1). During years of low activity, the PVL never received more than 5 submissions per week. During 1979 and 1980, dry conditions, suppressed the mosquito vectors. In 1978, mosquito vector levels were high, but the virus activity was low.

In years of high virus activity, horse cases appear to occur randomly across the southern agricultural areas of the province, without any noticeable clustering. However, they do appear to be distributed along the major waterways (Figs. 4-7). One major focus appears to be the Red River Valley. Another major focus is near Dauphin, contiguous to Dauphin Lake. A smaller number of cases were also located along the Assiniboine River Valley from Virden to Portage la Prairie.

Cases marked outside the Manitoba boundary (Figs. 5, 8) indicate submission from Ontario and Saskatchewan referred to the PVL for reasons of convenience.

In 1981 more of the older horses were affected than in 1977 (Fig. 8). In 1977, 6 vaccinated horses had a WEE infection whereas, in 1981, there were 36. These were confirmed, active cases of WEE. Reasons for the vaccine failure are unknown. Five horses that were vaccinated in 1981 died. Whether they had been given 1 or 2 doses of vaccine is unknown. It is known that about 12,000 doses of combined WEE/EEE vaccine were sold in the province in 1981 (Neufeld, unpublished data). This means that 15 to 30%, may have been immunized, depending on whether 1 or 2 doses of vaccine were used.

In 1981, 13 samples of brain were received by the PVL. Histopathological lesions of non-suppurative encephalitis, neuronal necrosis, neuronophagia, gliosis, and occasional meningitis were seen in 6 cases. Three virus isolations were made by the CPL. All specimens submitted to the A.D.R.I. for rabies antigen detection were negative.

The infection rate, in each crop district for the 3 outbreak years is given (Figs. 9-11). The highest infection rates were in districts 6, 7, 9, 11 and 12. These are the areas surrounding Dauphin, Portage la Prairie, Winnipeg-Steinbach, Winnipeg-Selkirk, and the Interlake region, respectively. Crop district 9, which includes the Eastern side of the Red River Valley consistently had the highest infection rate in all outbreak years.

During the 3 WEE outbreaks the average infection rate ranged from 2.3 to 4.6 cases per 1,000 horses; however, even though a high percentage of total cases came from districts 1, 2 and 3, their infection rates were lower than the eastern district which appears to be the main locus of WEE in Manitoba.

Vaccines

The vaccine which is used to immunize healthy horses, is produced by 3 companies[1]. It is believed to consist of formalin-inactivated virus, and to contain both WEE and EEE antigens and perhaps other products as well (i.e.; tetanus toxoid), but company policy prevents release of detailed information.

Immunization is accomplished by giving the recommended 2 doses at 10-14 day intervals. Annual re-vaccination is necessary to maintain protective levels of circulating antibody. It is customary practice in the field to use only one dose at the annual re-vaccination time as a booster for a horse which previously had received 2 doses.

Protection appears to be short-lived regardless of the procedure used. Experience in measuring antibody levels during outbreak years by the PVL indicated that many vaccinated horses have a titre of 1:32 or less by the HI test. In horses known to be adequately vaccinated, seroconversion occurs when they are infected and titres rise to levels of 1:4096 or greater (Neufeld, unpublished data). The CF titres in these horses may rise to 1:10, confirming a recent exposure to the virus (Lillie _et al_ 1976).

Some horses develop CNS disease even though they may have been vaccinated. Although 5 vaccinated horses died in 1981, our general impression from a review of case histories is that vaccinated horses suffer less severe manifestations of disease and recover more rapidly than unvaccinated ones.

DISCUSSION

The horse is a good sentinel animal for the detection of WEE virus transmission in the field. In the 1981 census, there were at least 31,284 horses in Manitoba. The number is actually higher than this because pleasure and race horses in suburban localities are not counted during the census. The actual population is estimated to be approximately 36,000.

There is a very definite difference in numbers of confirmed horse cases between outbreak and non-outbreak years. In non-outbreak years, confirmed cases never exceeded 10. During epizootic years, there were always more than 50 confirmed cases. If clinical cases of WEE exceed 6 per week in 2 consecutive weeks, we believe an epizootic will probably develop as occurred, in 1975, 1977 and 1981. A developing epizootic could be arrested by cold weather.

WEE infections occurred widely throughout the agricultural area of

---

[1] Cutter Laboratories, Shawnee, Kansas
Colorado Serum Company, Denver, Colorado
Fort Dodge Laboratories, Fort Dodge, Iowa

Southern Manitoba. Some clustering of horse cases occurred, but it is not known whether this was due to high local horse populations or to concentrations of virus-carrying mosquitoes. The infection rate indicates that there are high risk areas. This information could be used to decide which areas are best to monitor.

The equine deaths due to WEE in 1981 were substantial, including some vaccinated horses. These horses may not have been properly vaccinated. Young horses, especially those under one year of age, generally are more susceptible to the disease. Natural immunity due to previous exposure may protect many of the older horses.

The use of "sentinel farms" has been advocated by some workers. On such farms the WEE antibody of horses would be periodically monitored. Any rise in antibody titre would indicate a new exposure to WEE virus. Even vaccinated horses might serve as sentinels.

The use of the horse population in Manitoba as a surveillance tool has been valuable in the past and will continue to be so into the future. Even vaccination of horses will not negate their usefulness as sentinels. The identification of high risk areas, using infection rates may aid decision-makers in planning emergency spray programs. An early rise in horse cases may be used to predict an incipient outbreak in humans.

Acknowledgements

The assistance and cooperation of the veterinary practitioners in Manitoba is greatly appreciated. Technical assistance was provided by numerous laboratory staff and university students. Mr. John Giardino helped with graphs and tables.

References Cited

Lillie, L, Wong, F.C. and Drysdale, R.A. 1976. Equine Epizootic of Western Encephalomyelitis in Manitoba - 1975. Can. J. Public Health. 67(Suppl. 1):21-27.

Sekla, L.H. and Stackiw, W. 1976. Laboratory Diagnosis of Western Encephalomyelitis. Can. J. Public Health . 67(Suppl. 1):33-39.

Table 1

Manitoba Veterinary Medical Laboratory
Recorded Submissions from Horses With Western
Equine Encephalomyelitis - 1975 to 1981

Year	Total Submitted	Confirmed WEE	Percentage	Deaths Due to WEE[c]
1975[a]	261	139	53.3	6
1976	19	10	52.6	--
1977[a]	80	53	66.3	4
1978	5	1	20.0	--
1979	6	0	--	--
1980	11	1	9.0	--
1981[a]	187	128[b]	68.4	27

a. Highly significant infection rate compared to other years.

b. Confirmed cases from out of province (2 Saskatchewan, 3 Ontario) are included.

c. Laboratory confirmed cases. Many more horses reportedly died, but no specimens were submitted.

Figure 1

Crop districts in the southern agricultural area of Manitoba and the equine population distribution according to 1981 census figures

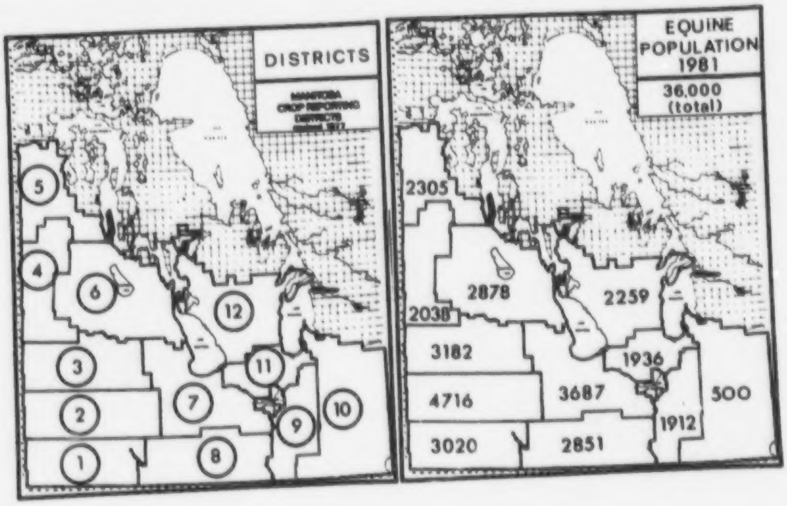

DISTRICTS
MANITOBA
CROP REPORTING
DISTRICTS
5
4
6
12
3
11
2
7
10
9
1
8
EQUINE
POPULATION
1981
36,000
(total)
2305
2878
2259
2038
3182
1936
4716
3687
500
1912
3020
2851

Figure 2

Equine population fluctuations on farms in Manitoba from 1922 to 1981 based on Statistic Canada census figures, Manitoba Department of Agriculture, Annual Reports.

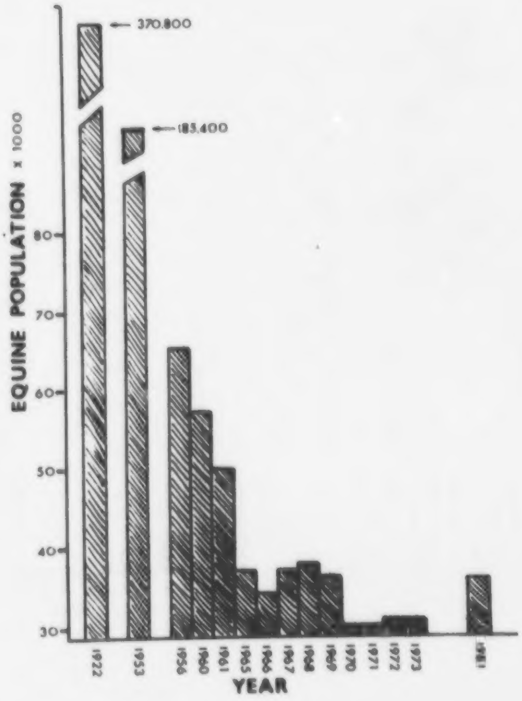


Figure 3

Number of confirmed horse cases of WEE according to age grouping; all horses less than 1 year old being included in the 1 year category.

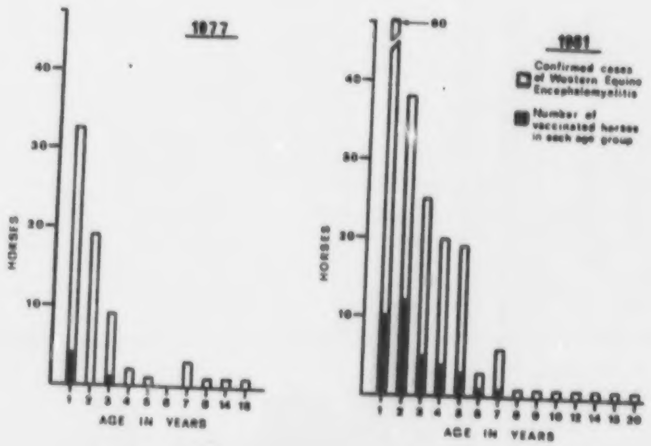


Figure 4

Total number of suspect WEE cases submitted to the Provincial Veterinary Laboratory per week during the three epizootic years.

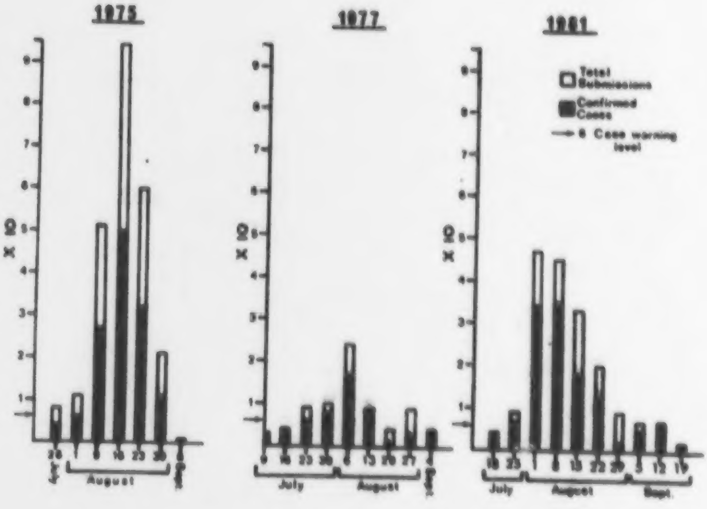


Figure 5

Distribution of clinical cases of WEE grouped according to municipality, in the southern agricultural area of Manitoba in 1975.

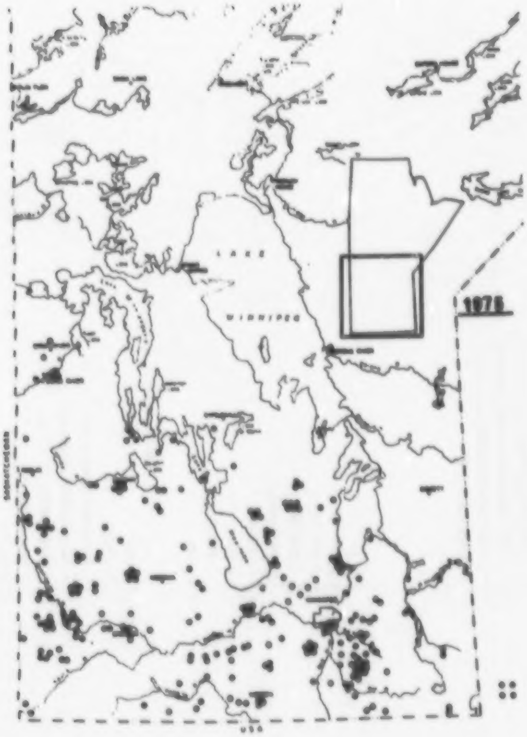

LAKE
WINNIPEG
1975
U.S.A.

Figure 6

Distribution of confirmed equine cases of WEE in Manitoba in 1976.

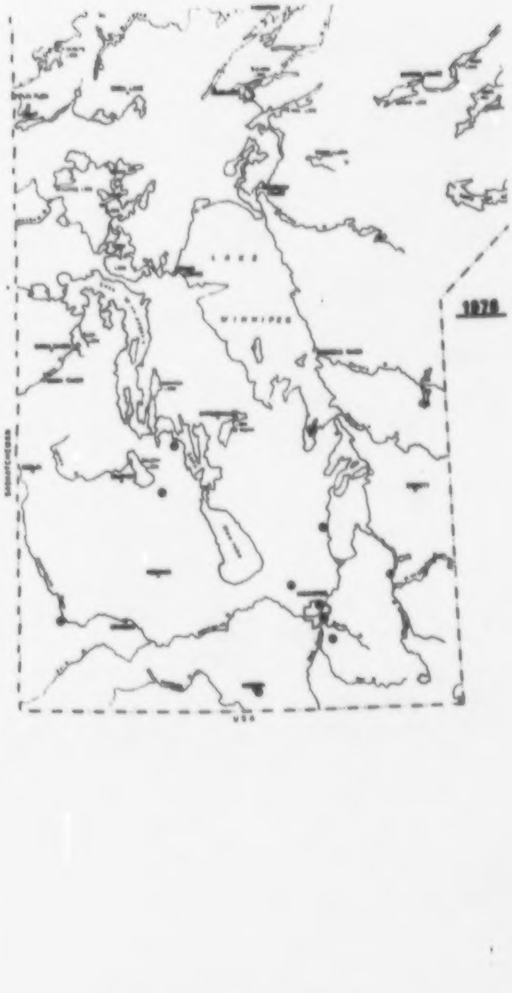

LAKE
WINNIPEG
1976

Figure 7

Distribution of confirmed equine cases of WEE in Manitoba in 1977.

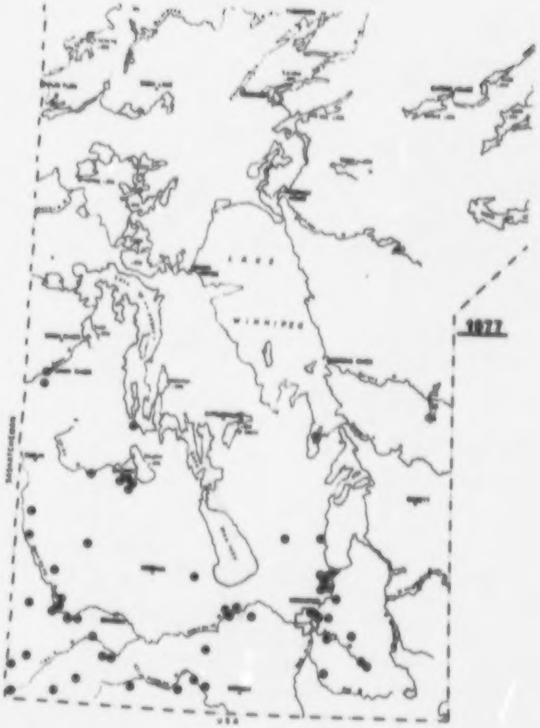

LAKE
WINNIPEG
1977

Figure 8

Distribution of confirmed equine cases of WEE in Manitoba in 1981.

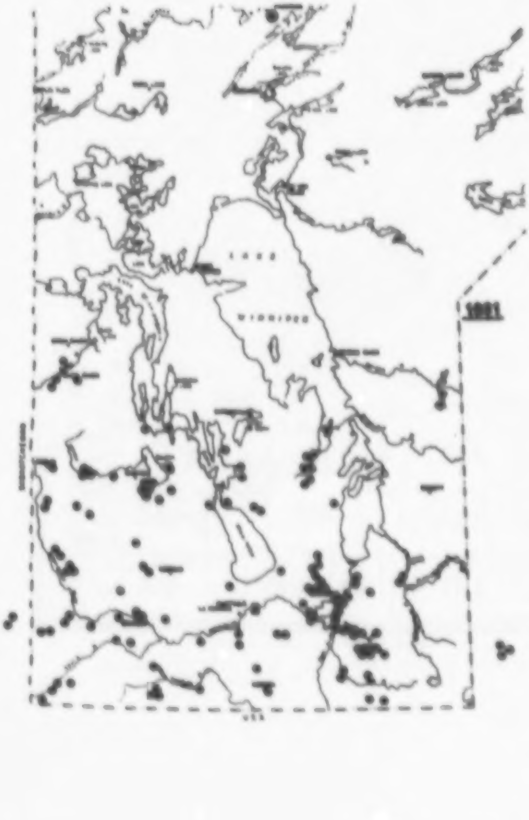

LAKE
WINNIPEG
1981
USA

Figure 9

Infection rate calculated per 1000 horses for each crop district for 1975.

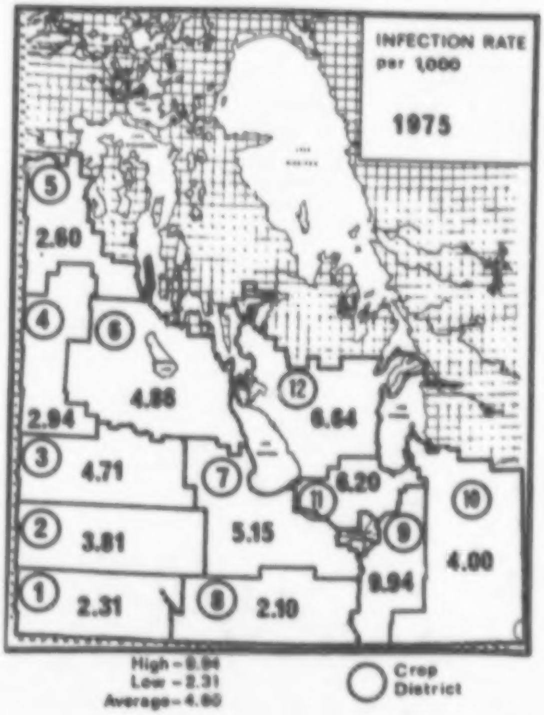

INFECTION RATE per 1,000
1975
5
2.60
4
2.94
6
4.86
12
6.64
3
4.71
7
5.15
11
6.20
10
4.00
2
3.81
9
9.94
1
2.31
8
2.10
High - 9.94
Low - 2.31
Average - 4.80
Crop District

Figure 10

Infection rate calculated per 1000 horses for each crop district for 1977.

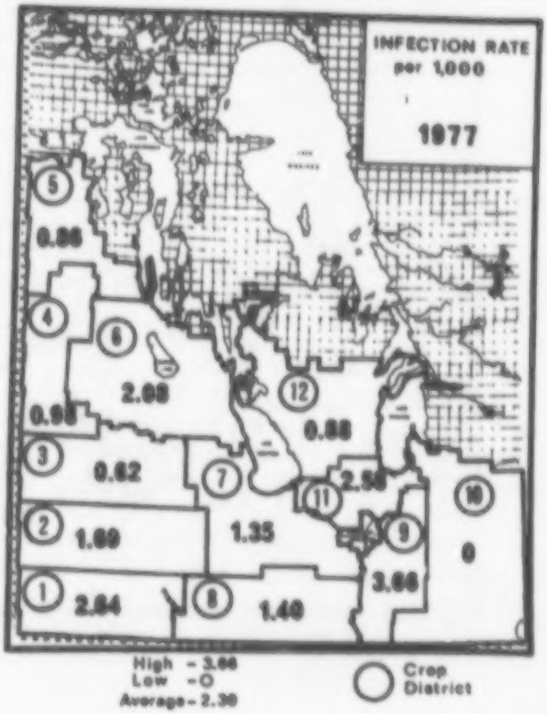
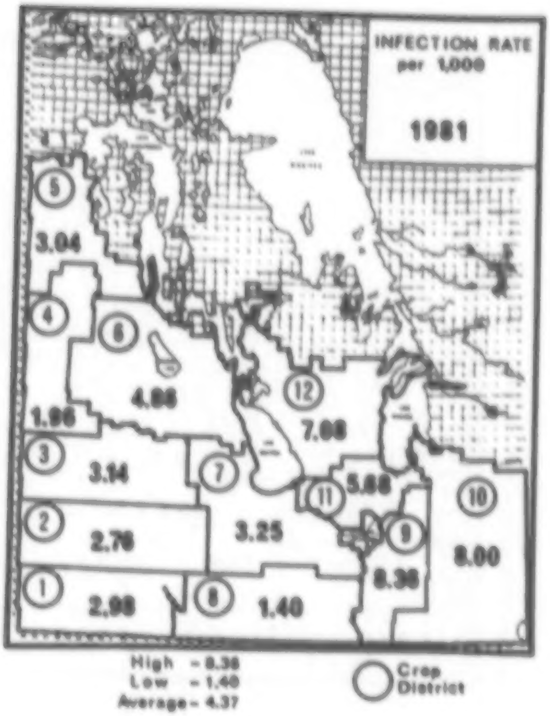

INFECTION RATE
per 1,000
1977
5
0.86
4
0.96
6
2.08
12
0.88
3
0.62
7
11
2.58
10
2
1.69
1.35
9
0
3.66
1
2.84
8
1.40
High - 3.66
Low - 0
Average - 2.30
Crop District

Figure 11

Infection rate calculated per 1000 horses for each crop district for the year, 1981.


INFECTION RATE
per 1,000
1981
5
3.04
4
1.96
6
4.86
12
7.08
3
3.14
7
11
5.68
10
2
2.76
3.25
9
8.00
8.36
1
2.98
8
1.40
High - 8.36
Low - 1.40
Average - 4.37
Crop District

Investigation for Western Equine Encephalitis Activity in Northwestern Ontario, 1981

G.A. Surgeoner, Ph.D.; W.E. Ralley, B.Sc.
Dept. Environmental Biology, University of Guelph, Guelph, Ontario, N1G 2W1
and M.S. Mahdy, M.P.H. D.Sc.
Arboviruses & Special Pathogens Unit, Virus Laboratory, Laboratory Services Branch, Ontario Ministry of Health, Box 9000, Terminal A, Toronto, Ontario, M5W 1R5.

ABSTRACT

During the 1981 western equine encephalitis (WEE) outbreak in Manitoba, a presumptive and a questionable horse case of the disease were reported in Fort Francis. Another case was later confirmed in Dryden. The three animals had no history of travel. A serological survey of 174 chickens from 14 flocks including those associated with the Fort Francis cases revealed no evidence of WEE antibodies. Adult mosquito trapping methods failed to collect Culex tarsalis although larvae were detected in Kenora and Fort Francis.

Historically a few isolated horse cases of WEE have been reported from northwestern Ontario (Roach, 1981). Two serologically presumptive positive and two presumptive negative cases in horses with demonstrated CNS abnormalities were also recorded during the 1975 outbreak in Manitoba (Lillie et al., 1976). There are no records to determine the extent of viral activity in bird populations or the potential species of vector mosquitoes in this region. The 1981 outbreak of WEE in Manitoba offered an unique opportunity to determine if the virus and its principal vector Culex tarsalis coq. represented a significant health threat to the residents of northwestern Ontario. Although less than 250 km from prairie regions of Winnipeg, the topography and vegetation of northwestern Ontario are drastically different. The area consists of primarily boreal forest vegetation and terrain typical of the Canadian Shield.

The objectives of this study were as follows: 1) To conduct a serological survey of chickens to investigate the possibility and extent of WEE activity relative to that in Manitoba; and 2) To determine relative numbers and species of mosquitoes from northwestern Ontario during mid-August; particularly the potential vector species.

MATERIALS AND METHODS

Samples were collected in this survey during the period from 12-15 August, 1981 in the regions of Kenora and Fort Francis, Ontario. The Dryden case occurred in September, samples from that area therefore were not collected.

Serology

Veterinarians in northwestern Ontario were notified by telephone of the ecological survey. With their cooperation, we identified individuals with small chicken flocks which were readily exposed to mosquitoes and obtained permission to bleed their birds. At each site the age of the birds and breed were recorded. Approximately 2 cc of blood were drawn from the wing vein using 10 cc vacutainer(R) test tube with a 1.5 inch 23 gauge needle. The tubes were labelled and stored in an ice chest maintained at approximately 5°C with ice packs.

Serological Testing

Sera were tested for WEE, St. Louis encephalitis and eastern equine encephalitis by the haemaglutination inhibition method. All sera were tested against the WEE (Manitoba) and Highland J antigens of WEE complex. In addition, chicken sera from the 2 farms where the presumptive and the questionable horse cases were reported, were tested using antigen prepared from the Flemming strain of WEE.

Mosquito Collection

Adult mosquitoes were aspirated in the early morning from the inside of chicken coops. The majority had blood-fed the previous night. These mosquitoes were considered representative of those feeding on ground-inhabiting birds. If chickens were shown to be seropositive to WEE, this could provide an indication of the likely vector species. Adults were also collected during 15 minute intervals as they landed on the forearms and front of human subjects. Counts were done in the Kenora and Fort Francis areas beginning ca. 9:15 pm and continued until ca. 9:45 pm, local time. Mosquitoes collected using this technique would be indicative of those species feeding on man. Mosquitoes were collected using New Jersey light traps. Three traps provided by the City of Winnipeg used 25 watt light bulbs as an attractant and 1 trap from the University of Manitoba used a 100 watt bulb. These traps were used to compare mosquito populations in northwestern Ontario to those in Manitoba. New Jersey light traps have been the basis of mosquito population estimates in the Manitoba outbreak. In addition, CDC light traps baited with dry ice were used to capture adult mosquitoes. These light traps were suspended from the tops of chicken coops. This trapping method is similar to that used for mosquito monitoring in the Essex Co. area of southwestern Ontario (Helson et al., 1980) and allowed for population comparisons between southwestern Ontario and northwestern Ontario.

While visiting farms all potential mosquito breeding sites were checked for larvae, pupae and egg rafts to determine which species of mosquitoes were breeding in the area.

RESULTS

Serological results are presented in Table I. There was no evidence of WEE antibodies in any of the chickens tested. One white leghorn from the Emo region had an HAI titre of 40 to EEE antigen. The reaction was most likely non-specific since there was neither local nor contiguous EEE activity. This serum has not been tested for neutralizing antibodies. Mosquito trapping results are presented in Table II. *Culiseta inornata* and *A. nigromaculis* were collected from the Fort Francis area only; other species were found in both localities. Adults of *Culex tarsalis* were not captured although larvae were found in a roadside ditch in Kenora and in a cesspool on a farm in the Fort Francis region.

DISCUSSION

Veterinarians and horse owners estimated the horse population to be about 900 animals, 600 in Fort Francis area and 300 in Kenora. Sixty percent of these horses would be vaccinated against WEE. There were two horse cases from northwestern Ontario, one presumptive in the Fort Francis region and one confirmed in the Dryden area. Although numbers are so small and comparisons thus questionable, the estimate of WEE in horses in northwestern Ontario is low compared to Manitoba.

There was no evidence of widespread WEE activity based on serological survey of chickens. At the time of this survey most sentinel flocks in Manitoba had shown greater than 50% antibody conversion to WEE. Adults of *Culex tarsalis* the primary vector of WEE virus were not captured; although the species was present based on larval collections. The low numbers of *C. tarsalis* may explain the lack of WEE activity in chickens. Perhaps a different epidemiological cycle for WEE involving different mosquito species is occurring in the region. The questionable horse case was reported from the Fort Francis area with symptoms in the animal developing about June 15. *Culex restuans* and *Culiseta inornata*, potential secondary vectors of WEE, were captured in low numbers. Population levels were slightly lower than those of Manitoba. At the onset of illness, this animal was vaccinated against WEE. Validity of the serological results is clouded and the case is therefore questionable. The major species of mosquitoes feeding on chickens were *Mansonia perturbans* and *Anopheles walkeri*, both of which develop in permanent bodies of water where macrophyte vegetation exists. *M. perturbans* may be a potential vector of WEE (Sekla et al., 1980). As in Manitoba, the most common species feeding on man was *A. vexans* (Table II).

In northwestern Ontario; Kenora, Fort Francis, Rainy River and Dryden are the only major municipalities. There is however a large scattered rural and tourist population. More epidemiological information is required to determine the need for vector control. However, based on the lack of antibody activity in chickens and the low incidence of *C. tarsalis*, vector control would not be justified using criteria currently used in Manitoba.

Acknowledgments

This survey was funded by the Ontario Ministry of Health. We would like to thank the veterinarians and chicken owners in northwestern Ontario who provided complete cooperation and made the program possible. The assistance from Dr. R. Ellis, City of Winnipeg and Dr. R. Brust, University of Manitoba is gratefully acknowledged.

References Cited

Helson, B.V. et al. 1980. The seasonal distribution and species distribution and species composition of mosquitoes collected in a St. Louis encephalitis surveillance program for 1976-78 in southwestern Ontario. Can. Ent. 112: 865-874.

Lillie, L.E. et al. 1976. Equine epizootic of western encephalomyelitis in Manitoba - 1975. Can. J P H. Supp. to 67: 21, 1976.

Roach, W. 1981. Northwestern Ontario MDs on Alert as Encephalitis Confirmed in Two Horses. Toronto Globe and Mail. August 7, 1981.

Sekla, L.H., W. Stackiw and R.A. Brust. 1980. Arbovirus isolations from mosquitoes in Manitoba. Mosq. News 40: 377-380.

Table I. Serological results from chicken sera, northwestern Ontario, 1981.

Location	No. of Flocks Tested	No. of Birds Tested	Seropositive[1]
Kenora	4	50	None
Devlin	3	31[2]	None
Emo	4	50	1 ± to EEE[4]
Stratton	3	43[3]	None
Total	14	174	

[1] Sera were tested against antigens from 3 strains of western equine encephalitis complex, eastern equine (EEE) and St. Louis encephalitis (SLE) antigens.

[2] Including 6 chickens from farm on which questionable horse case occurred.

[3] Including 21 chickens from farm on which presumptive horse case occurred.

[4] HAI of 40 to EEE antigen only, probably non-specific.

Table II. Mosquito species collected using various collecting methods, northwestern Ontario, August 12-15, 1981.

Species	A/C[1]	CDC[2]	N.J.[3]	H/B[4]	Larvae[5]
Mansonia perturbans	58	141	55	14	
Aedes vexans	7	121	15	33	+
Aedes canadensis	3	4	3	-	
Aedes spencerii	1	2	-	5	
Aedes communis	-	3	3	9	
Aedes nigromaculis	-	-	1	-	
Aedes spp.	-	20	12	1	
Anopheles walkeri	28	4	4	5	
Anopheles earlei	8	6	3	1	
Culiseta inornata	-	4	1	-	+
Culex restuans	6	8	4	-	+
Culex tarsalis	-	-	-	-	+

[1] Adults aspirated from 8 chicken coops in early morning.

[2] Based on collections from 5 CDC traps baited with dry ice.

[3] Based on collections from 8 New Jersey light traps.

[4] Based on 6 evening human biting collections.

[5] *Culiseta inornata*, *Culex restuans* and *Culex tarsalis* all found at site of 1 presumptive WEE horse case.

CHAPTER IV:

Sentinel Chicken Flocks Data

Sentinel Flock Monitoring Procedure for Western Equine Encephalomyelitis in Manitoba 1976-1981

F.C. Wong Ph.D.
J.L. Neufeld D.V.M., Diplomate A.C.V.P.

Veterinary Services Branch
Manitoba Department of Agriculture
545 University Crescent
Winnipeg, Manitoba R3T 2N2

Abstract

Surveillance to detect activity of Western Equine Encephalitis (WEE) has been carried out in Manitoba since 1975. Some basic changes in the program have occurred: strengthened cages, addition of a mosquito live-trap to the flock cage, use of laying birds instead of broilers, and repeated bleeding in the same birds left in place for the duration of the testing period (summer). During the epizootics of 1977 and 1981, the percentage of positive birds quickly surpassed 20 in the majority of sites. In 1977, there were 135 positive out of a total of 830 birds tested. In non-epidemic years the percentage of positive birds ranged from 0.74 to 3.30. In 1981, with sites reduced to 5, and birds in place all summer, 63 out of a possible 75 seroconverted (84.0%). In conclusion, it is suggested that a small number of sites (minimum 6) are adequate to predict an incipient outbreak of WEE, and surveillance should be continued consistently at this minimum level every year to generate baseline data from non-active years for comparison. Positive birds are defined as those which seroconvert to the 1:32 level or greater by the HI test. If the rate of seroconversion in sentinel flocks surpassed 20% at any one bleeding, an epizootic in horses and an epidemic in the human population will occur.

Since 1975, when an outbreak of Western Equine Encephalomyelitis (WEE) was documented in Manitoba (Waters 1976), an annual sentinel flock surveillance program has been instituted every summer. This paper reports the results of the annual surveillance from 1976-1981, together with the modifications made to the basic procedures previously described (Wong 1976), and our recommendations for future monitoring methods.

MATERIALS AND METHODS

Sentinel Birds

From 1976 to 1979, 5 week old commercial broiler chickens of the Cobb Strain were used as previously reported (Wong 1976). For 1980 and 1981, the broilers were replaced with "brown layers", a strain derived from the Rhode Island Red. One-day old birds were obtained from a commercial source[1] and were raised indoors, free from mosquito exposure. The birds were shown to be serologically negative to WEE. No vaccinations or medications were administered. The birds were 5 weeks old with an average weight of approximately 1.0 Kg when they were put out on location.

During the surveillance period, the birds were maintained on a complete 18% pelleted grower ration[2], medicated with Coyden-25[3]. Clotted blood samples were taken from these birds approximately every 2 weeks to test for seroconversion. Birds showing an haemagglutination-inhibition (HI) titre of 1:32 or higher were considered positive and were removed from the flock.

Cages

The basic design of the sentinel flock cage remained the same as previously described except for 2 major modifications. First a partition was added between the area bound by the wire mesh and wooden panel creating an area sheltered on all sides from wind or rain. This was done to decrease the effects of exposure on the birds during inclement weather, and to provide an overnight resting place. Second, in certain locations, where distance allowed daily collection, a mosquito live-trap was attached to the cage (Figure 1). It should also be noted that the cage frame and the wire required strengthening, hence the replacement by a heavier 25 mm x 50 mm x 16 gauge mesh. The mosquito live-trap was attached to the wooden end of the cage and trapped mosquitoes as they left after taking their nocturnal bloodmeals.

Flock Locations

Criteria used in choosing the flock locations have been described (Wong et al 1976). However, the number of flocks year to year is governed by budget and available resources. Each year, a flock was consistently located in each of 4 sites; i.e., Winnipeg (designated as Charleswood), Portage la Prairie, Brandon, and Oak Hammock. Between 1976 and 1981, a total of 21 flock sites were located in the 12 crop districts (Figure 2). In 1976, one flock was located in Thompson, 755 km north of Winnipeg. In 1981, the number of flocks was reduced to 5. Because comparison between years is important, the 4 sites maintained on a year to year basis without interruption have been used in the presentation of data before. The total number of sites used in any one year is shown in Table 1.

1/ Miller's Hatchery, 260 Main Street, Winnipeg, Manitoba, R3C 1A9

2/ Maple Leaf Mills Ltd. 440 Archibald Street, Winnipeg, Manitoba R2J 0X4

3/ Coyden-25 (25% clopidol), Agriculture Products Development, Dow Chemicals of Canada Ltd., Sarnia, Ontario.

Flock, Scheduling and Bleeding

Between 1976 and 1979, the flock replacement method was continued as previously reported (Wong et al 1976). There were 4 exposure periods. The first was between May 15 and June 7-14. The second ended by July 4-9 followed by the third ending July 25-August 4. The fourth terminated by August 14-27.

In 1980 and 1981, the repeat bleeding method was used. Brown layers were chosen for 2 reasons, i.e.; their blood samples yield a higher proportion of serum per unit volume of clotted blood and their smaller size alleviates the over-crowding problem. A pre-exposure sample consisting of 5 ml of blood was removed from the deep ulnar (wing) vein of each bird and allowed to clot. The first post-exposure samples was taken about 30 days after the birds had been on location, and thereafter a sample was taken at least every 2 weeks (or more frequently, if evidence of activity apparent). Each exposure samples consisted of 4-5 ml of clotted blood.

Serology

The blood serum was harvested from the clotted sample after centrifugation and was tested using HI procedures (Sekla and Stackiw 1976). As noted above, a HI titre of 1:32 or higher was considered a seroconversion. A 20% seroconversion per flock per exposure period indicates high viral activity in the mosquito vector.

RESULTS

By the flock replacement method, the results of the 4 permanent sites for the period between 1976 and 1979 are shown (Table 2). In 1977, all 4 locations showed a greater than 20% seroconversion by the third cycle ending by August. Similar results were observed in 8 of the other 12 locations (Table 3). The HI titres ranged from 1:256 to 1:1024 or higher. Based on the information generated from the sentinel flocks, the high incidence of the vector, the distribution of equine cases WEE, and WEE virus isolation from mosquitoes in the flock traps, an outbreak of WEE was predicted.

In 1980, the percentage of seroconversion for each sampling period was calculated on the number of negative birds which turned positive after each sampling period. For the period of May 15 to August 28, a total of 743 blood samples were collected from chickens placed in 10 locations; only 5 birds showed seroconversion.

In 1981, the number of flock locations was reduced to 5; i.e.; the 4 permanent sites, and Glenlea. In 4 flocks seroconversion ranged from 33% to 64% by July 21. Between July 21 and 28, the remaining flock seroconverted to 100% from its previous level of 13% on July 21 at the Brandon site (Table 4).

In non-epidemic years, the number of sentinel chickens which seroconverted was extremely low. In 1976, when 20 flocks were in operation, only 8 birds seroconverted out of a total of 1080. In 1978, from 10 locations, 19 birds seroconverted out of a total of 578 birds. In 1980, 5 birds seroconverted from a total of 150 chickens, (Table 1).

DISCUSSION

In a WEE endemic area, sentinel flocks have proven to be a reliable means of monitoring viral activity. The large difference between the percentage of positive birds in epidemic years and that of non-epidemic years requires no statistical analysis to ascertain its significance (Table 1).

The repeat bleeding method increased the sensitivity of the viral activity surveillance. In 1977, the flock replacement method produced 16.26% positive birds only, whereas the repeat bleeding method produced 84% positive chickens in 1981.

Using the repeat bleeding method, one could question whether or not the seroconversion was higher because of multiple feedings of mosquitoes who have or may have had their first blood meal on a viremic bird. Such a multiple feeding in one gonotrophic cycle is not considered likely to occur because mosquitoes feed on the sleeping chickens after sunset, when the birds are defenseless against the completion of a bloodmeal. Previous studies have shown that the maximum duration of WEE viremia in domestic chickens is a maximum period of 3 days (Wong et al 1976).

Sentinel flocks in the 12 crop districts have yeilded fairly uniform results. When no viral activity was observed, the percentage of seroconversion of birds was low, irrespective of the number of flocks in the province. For example, in 1976, a non-epidemic year, 20 flocks yielded 0.74% positive birds. The efficacy of even a few sites was substantiated in 1981 when only 5 flocks were used. This small number of flocks was adequate to generate sufficient data to forecast a WEE outbreak which developed throughout Southern Manitoba (Neufeld, in press).

The uniformity of the viral activity is attributed to the similarity of weather conditions throughout the 12 crop districts, especially with respect to temperature and rainfall. There also exists some similarity in the distribution of natural mosquito habitats. Therefore, it may be possible to monitor mosquito activity in a few selected sites and to apply the results to a wider area through experience and knowledge of previous outbreaks. The results are easy to confirm using horses as a sensistive surveillance monitor. Horse cases tend to occur 1 or 2 weeks after the first detection of seroconversion in the sentinel chicken flocks.

Initially, it was planned to replace a positive bird with a fresh negative bird to maintain a constant number of 15 per flock in the repeat bleeding method. However, our experience in 1981 showed that the replacement or negative bird had a limited value because it took 7 days to replace a positive bird at a site after blood had been taken and tested and because the replacement bird did not yield its first 7 days' exposure sample until 14 days after the bleeding of the original seroconversion. The value of serological data from such a short exposure is questionable, especially because it has been determined that 7 days following the injection of a large dose of WEE virus the seroconversion is at a low level (Wong et al 1976).

The addition of the negative bird(s) in replacement of a positive bird(s) introduces a dilution factor that could seriously alter the rate of seroconversion during any one sampling period. Because statistical analysis cannot accommodate this variable, the seroconversion rate was computed according to the number of birds remaining at the site at each bleeding. The accumulated results for the 5 sites used in 1981 are shown (Table 4).

The method of repeat bleeding has one obvious fault. For example, at the Brandon site, all birds may seroconvert over a short period of time. As a result of high viral activity in the mosquito vector, this effectively removes that site from the surveillance system for the length of time it takes to set up a new sentinel flock. The new birds cannot provide any useful data for at least 2 weeks.

This is not a problem for an early-warning system to monitor WEE activity. However, it is difficult to study the dynamics of viral activity on a seasonal basis.

CONCLUSIONS

Monitoring WEE vector activity using sentinel flocks has proven to be of invaluable in predicting incipient epizootics in horses and epidemics in humans over the past 7 years. The addition of mosquito livetraps to the flock cages has contributed to the overall assessment of WEE activity.

Permanent flock sites are the only means by which meaningful comparative data can be generated. Also, repeated weekly or bi-weekly blood sampling from the flocks is more sensitive than total flock replacement at 2 week intervals. The use of "brown layers" chickens reduced the overcrowding problem associated with commercial broilers. Because viral activity in sentinel chickens has never been detected earlier than June 1, it may not be necessary to place surveillance flocks on the site until the 15th of May.

The sentinel flock program generates useful comparative data only if it is operated on a permanent, year by year basis, and at the same surveillance sites. If the sites were permanent, the sentinel flocks might prove useful in assessing the efficacy.

The 4 sites which have consistently been used between 1975 and 1981, provide adequate predictive data, but the number should be increased to 6 sites. The additional 2 sites are needed within the built up area of Winnipeg to indicate virus activity in the vectors which occur in this densely populated area. At the present time the only data gathered within the City are mosquito counts from the New Jersey light traps.

The sites at Portage la Prairie, Brandon, and Oak Hammock should be located away from population centres, so that they could serve as "no spray" controls, in the event that adulticiding is carried out during an emergency. Only in this way will information be generated which can be used in assessing efficacy of large-scale adulticiding spray programs. However, this would mean the loss of whatever comparative data is available.

An incipient outbreak of WEE is probable when a sentinel flock seroconversion rate exceeds 20%. A outbreak is in full progress if the percentage of seroconversion increases at the next bleeding cycle. These warning parameters have been developed during 3 serious epizootics and might be used with confidence in any climatic or geographical area which approximates those conditions found in Manitoba.

Acknowledgements

The authors acknowledge the support of the several rural veterinary practitioners who assisted with sentinel flocks. Numerous students from the University of Manitoba assisted during the summer months, 1976-1981. Mrs. Gayle Anderson and Mrs. Donalda Bowness provided valuable technical assistance, Mr. John Giardino provided help and produced the illustrations.

References Cited

Sekla, L.H. and Stackiw, W. 1976. Laboratory diagnosis of Western Encephalomyelitis. Can. J. Public Health. (Supplement 1) 67:33-39.

Waters, J. 1976. An epidemic of Western Encephalomyelitis in humans - Manitoba, 1975. Can. J. Public Health (Supplement 1) 67:28-32.

Wong, F.C., Lillie, L., Drysdale, R. 1976. Sentinel flock monitoring procedures for Western Encephalomyelitis in Manitoba - 1975. Can. J. Public Health (Supplement 1) 67:15-20.

Table 1

Seroconversion to Western Encephalomyelitis Virus in Sentinel Chickens in Manitoba, 1975 - 1981

Year	No. of Locations	No. of Birds Used	No. of Sero-Positive Birds	Percentage of Positive Birds
1975[1]	11	820	214	26.1
1976	20	1080	8	0.74
1977[1]	15	830	135	16.26
1978	10	578	19	3.28
1979	10	561	6	1.07
1980	10	150	5	3.30
1981[1]	5	75	63	84.0

1/ Epidemic years

Table 2

Percent Seroconversion of Sentinel Chickens from 4 Permanent Sites, 1976 - 1981

Year	Period	Charleswood	Portage	Brandon	Oak Hammock
1976	1	0	0	0	No operation
	2	14	0	13	No operation
	3	0	21	0	No operation
	4	0	0	0	No operation
1977	1	0	0	0	0
	2	0	0	0	0
	3	60	93	43	64
	4	0	40	50	0
1978	1	0	0	0	0
	2	0	0	0	6.6
	3	0	0	6.6	0
	4	7.7	7.7	6.6	16
1979	1	0	0	0	0
	2	0	0	0	0
	3	0	0	0	0
	4	0	8	9	9
1980	1	0	0	0	0
	2	0	0	0	0
	3	0	0	0	0
	4 - 6	0	0	0	0
1981	1 ending 10 June	0	0	0	0
	2 ending 23 June	0	0	0	0
	3 ending 14 July	6	Not done	0	0
	4 ending 21 July	64	33	13	40
	5 ending 28 July	0	30	100	44
	6 ending 5 Aug.	60	28	No birds	40

Table 3

Percent Seroconversion[a] of Sentinel Flocks in Manitoba 1977

		Period 1	Period 2	Period 3	Period 4
1.	Charleswood			60	
2.	Selkirk		6.6	84	13
3.	Morris			50	6.6
4.	Portage			93	40
5.	Brandon			43	50
6.	U. of M.			30	7
7.	Dauphin			71	27
8.	Virden			50	38
9.	Killarney		6.6	30	53
10.	Oak Hammock			64	
11.	Steinbach			17	
12.	Arborg			46	
13.	Morden	6.6		15	27
14.	Pinawa				
15.	Melita		6.6		43

Total Birds Tested - 830

Positive Birds by Seroconversion - 135

---

a = Interpretation of seroconversion - a rise in WEE antibody titre from less than 1:8, to a level of 1:32 or greater using the HI test.

Table 4

Seroconversion of Sentinel Flocks in Manitoba 1981[1]

Location	Period of Exposure	No.+/No. Tested	Percent Seroconverted
Glenlea	22 May to 9 June	0/15	0
	to 23 June	0/15	0
	to 14 July	1/15	6
	to 21 July	9/14	64
	to 28 July	0/5	0
	to 5 August	3/5	60
	Accumulative	13/15	86
Oak Hammock	22 May to 11 June	0/14	0
	to 25 June	0/15	0
	to 14 July	0/15	0
	to 21 July	6/15	40
	to 28 July	4/9	44
	to 5 August	2/5	40
	Accumulative	12/15	80
Charleswood	22 May to 9 June	0/15	0
	to 25 June	0/15	0
	to 14 July	1/15	6
	to 21 July	7/14	50
	to 28 July	4/7	57
	to 5 August	1/3	33
	Accumulative	13/15	86
Portage	22 May to 10 June	0/13	0
	to 24 June	0/15	0
	to 21 July	5/15	30
	to 27 July	3/10	33
	to 5 August	2/7	28
	Accumulative	10/15	66
Brandon	22 May to 11 June	0/15	0
	to 25 June	0/15	0
	to 14 July	0/15	0
	to 24 July	2/15	13
	to 30 July	13/13	100
	Accumulative	15/15	100

1. The data is cumulative to August 5 or July 30. At that time aerial spraying began and all birds were replaced.

Diagram of cages used in 1981 WEE sentinel flock surveillance program.
Dimensions are metric.

Figure 1

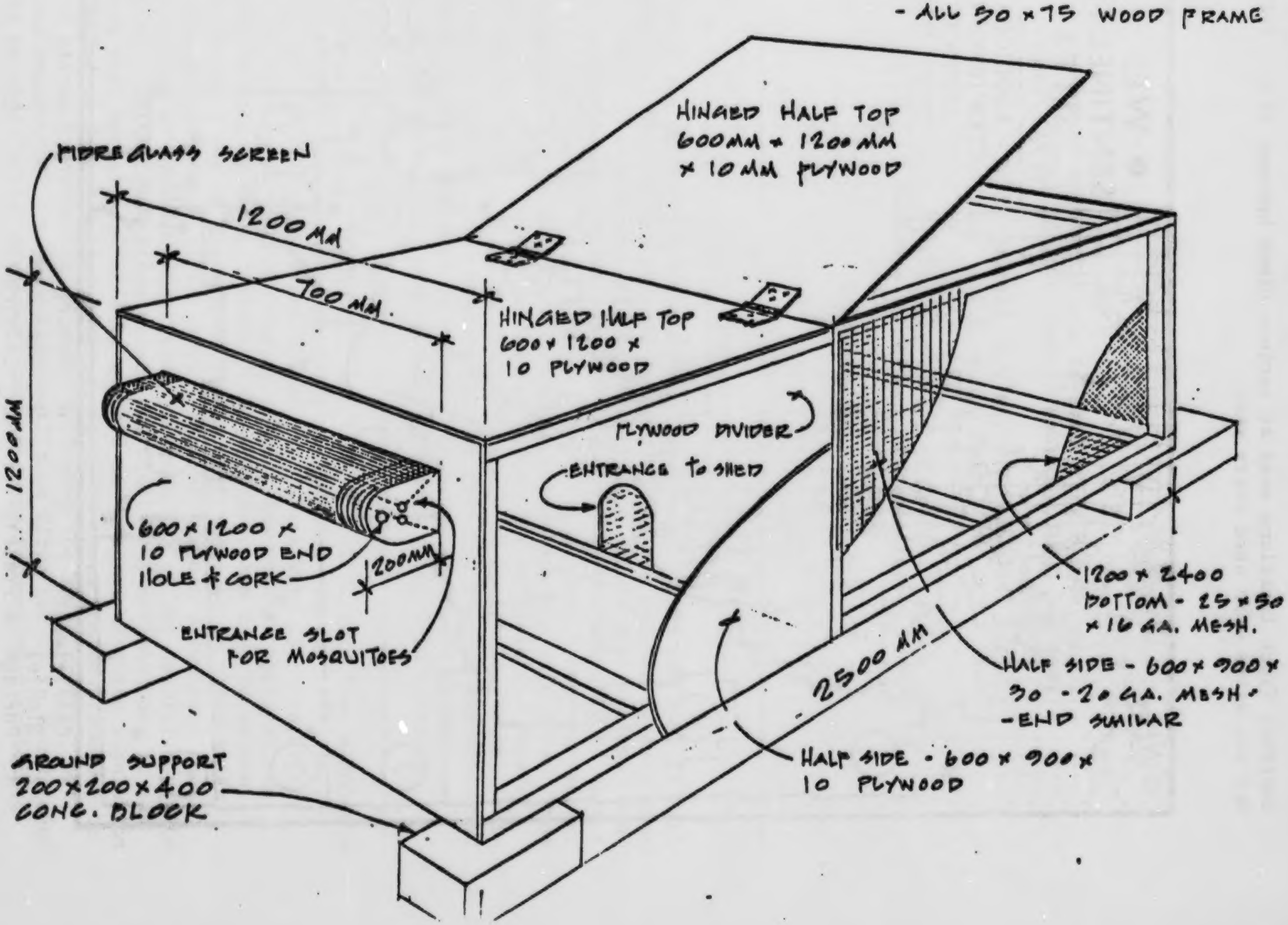
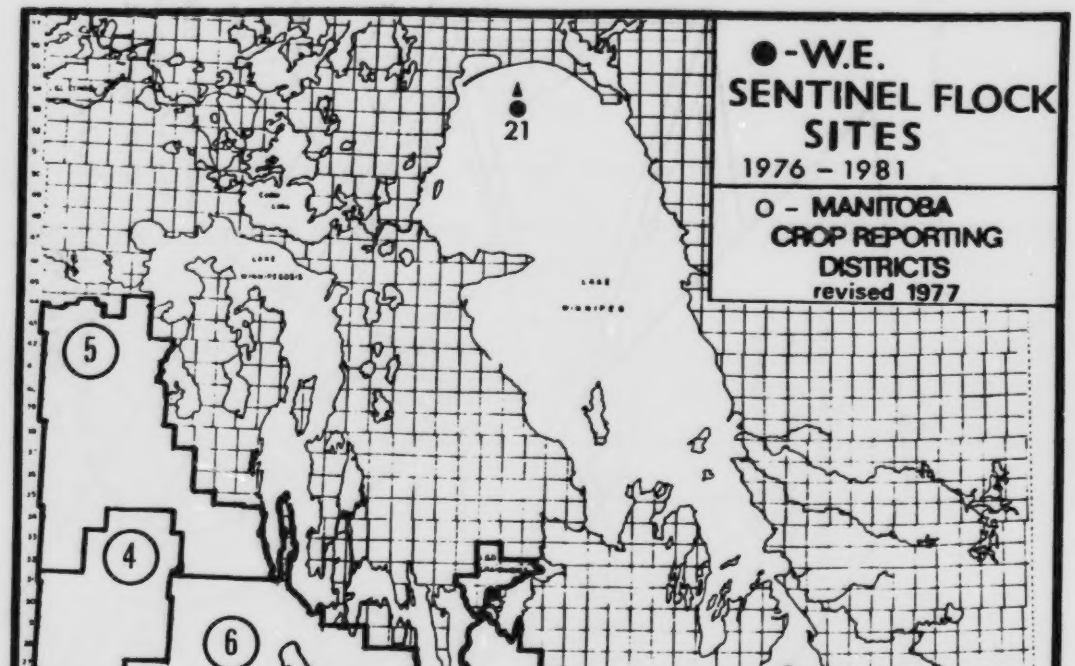

- ALL 50 x 75 WOOD FRAME
HINGED HALF TOP
600MM x 1200MM
x 10MM PLYWOOD
FIBREGLASS SCREEN
1200MM
700MM
HINGED HALF TOP
600 x 1200 x
10 PLYWOOD
1200MM
600 x 1200 x
10 PLYWOOD END
1HOLE & CORK
200MM
ENTRANCE SLOT
FOR MOSQUITOES
PLYWOOD DIVIDER
ENTRANCE TO SHED
1200 x 2400
BOTTOM - 25 x 50
x 16 GA. MESH.
2500 MM
HALF SIDE - 600 x 900 x
30 - 20 GA. MESH -
- END SIMILAR
HALF SIDE - 600 x 900 x
10 PLYWOOD
GROUND SUPPORT
200 x 200 x 400
CONC. BLOCK

Figure 2

Sentinel flock locations used at various times between 1976 - 1981
Not all sites were used every year.


●-W.E.
SENTINEL FLOCK
SITES
1976 - 1981
O - MANITOBA
CROP REPORTING
DISTRICTS
revised 1977

1. Charleswood
2. Glenlea
3. Portage la Prairie
4. Campus - U. of Man.
5. Brandon
6. Oak Hammock
7. Morden
8. Arborg
9. Steinbach
10. Selkirk
11. Morris
12. Pinawa
13. Virden
14. Killarney
15. Melita
16. Dauphin
17. Lac du Bonnet
18. Gimli
19. St. Pierre
20. Stonewall
21. Thompson

Use of Sentinel Birds in Evaluating Effectiveness of Aerial Adulticiding in Manitoba, 1977 and 1981

J.L. Neufeld, D.V.M., Diplomate A.C.V.P.
F.C. Wong, Ph.D.

Veterinary Services Branch
Manitoba Department of Agriculture
545 University Crescent
Winnipeg, Manitoba
R3T 2N2

Abstract

After the emergency vector control programs of 1977 and 1981 in Manitoba, sentinel chicken flocks were located in sprayed and non-sprayed areas in an attempt to assess the effectiveness of adulticide application. Conclusive data were not obtained in 1977 because cool weather stopped vector activity. In 1981, equivocal results were obtained from 4 non-sprayed and 10 sprayed sites, with minor indication that spraying was effective. Wide variations in results between sites were difficult to explain. However, sentinel flocks appear to be an effective means of monitoring vector activity, even in late August. Reasons for the variable results are discussed and recommendations are made to enhance the value of future attempts at monitoring the efficacy of adulticiding programs using this approach.

Between 1975 and 1981, 3 serious outbreaks of Western Equine Encephalomyelitis (WEE) have occurred in Manitoba, Canada. During 1975, 1977 and 1981, substantial numbers of horses and humans were affected by the disease (Lillie et al 1976 and Waters 1976). Insecticide applications were made in each of those 3 years. The only approach used to assess efficacy in 1975 was to place test groups of mosquitoes in those areas to be sprayed (Brust and Ellis 1976). The conclusions drawn from that study indicate an overall vector kill of about 80% of the natural mosquito population. Using land-biting counts and light-trap collections, it was estimated that aerial adulticiding reduced the matural mosquito population for a period of 3 to 7 days. However, in 1975, there was no procedure to evaluate vector transmission of virus following the spray program, nor was there any control group(s) of surveillance birds set up in non-sprayed areas to provide "normal activity" data in comparison to data collected in sprayed areas.

This paper described the procedures for setting up sentinel flocks in sprayed and non-sprayed areas, in an attempt to assess the effectiveness of aerial adulticiding programs.

MATERIALS AND METHODS

Sentinel birds, tested prior to placement to ensure they were negative for WEE antibody, were placed in the surveillance cages and maintained as described for the routine WEE surveillance (Wong and Neufeld, 1982). From 5 to 15 birds were placed in each location within 24 hours of the actual spraying of the spray block or population centre. For controls, some sentinel cages were placed in non-sprayed areas, at least several kilometers from the edge of a spray zone. Bleeding of birds was begun approximately 2 weeks following placement. Serum was harvested from clotted blood samples and tested using the haemagglutination-inhibition procedures as previously described (Sekla and Stackiw, 1976). Any positive bird with an antibody titre over 1:32 was removed.

In 1977, birds were placed near the following centres after aerial spraying with propoxur: Winnipeg, Portage la Prairie, Selkirk, Brandon, Dauphin, Virden and Morris. Control birds were placed at the same time in 7 other locations which had not received any insecticide spray: Oak Hammock, Morden, Arborg, Pinawa, Steinbach, Melita and Killarney. The difference in seroconversion rate between birds near sprayed centres and control birds was expected to indicate the effectiveness of spraying by showing a lower rate of seroconversion in sprayed areas. The post-spray exposure period was 17 days.

In 1981, a total of 21 communities (Ellis, 1982) were treated with aerial adulticiding agents. Fourteen flocks were set out in the areas indicated (Table 1). Fifteen birds were placed in each cage. Four of the 14 flocks were placed in unsprayed areas as controls. The exposure periods ranged from 11 to 40 days.

RESULTS

In 1977, none of the birds seroconverted, following spraying in any of the locations.

In 1981, the birds demonstrated a variable rate of seroconversion, from 0 to 66% (Table 1). In the control sites, the birds at Traverse Bay were negative, but those at Glenlea, Oak Hammock and at Arborg showed a 7%, 15% and 50% seroconversion, respectively. In 4 of the 10 sprayed centres, seroconversion was nil. In 2 centres, the seroconversion was 6 and 14%. In sites, seroconversion ranged from 21 - 66%; these were Selkirk, Portage la Prairie, Charleswood and Brandon.

DISCUSSION

In 1977, pre-spraying sentinel flock seroconversions, occurred during the first week of August. After an emergency was declared, spraying took place between August 14 and 17. New birds were put in place at this time.

However, because of subsequent low temperature vector activity was reduced (Ellis and Brust, 1982).

At 6 of the sprayed sites, seroconversion remained at or below 14%. At 4 of these, seroconversion was nil. This data suggests that adulticiding was effective. Because some flocks were left in place for rather lengthy time periods to detect return of victor/virus activity, the chance for them to seroconvert was enhanced. Those flocks with $\leq$ 14% seroconversion were generally left in place 2 to 3 weeks only. This would allow sufficient time for incubation of infection from the immediate post-spray time period, and would not expose the birds to a build-up of "new" virus-carrying vectors. In 4 sites, seroconversion rates were substantial. In Portage la Prairie, mosquito counts were lowered for up to 6 days, then rebounded. Because of the variables in exposure times following spraying, vector activity at different sites and presence or absence of virus and dates of aerial spraying, the results are inconclusive as to the effectiveness of aerial spraying. However, the approach to monitoring effectiveness shows promise and contingency plans should be prepared so that this method of assessment could be used in future spraying programs.

References Cited

Brust, R. and Ellis, R. 1976. Assessment of the Emergency Mosquito Control Operation in Manitoba, 1975. Can. J. Public Health (Suppl.) 67:60-71.

Ellis, R. 1982. Emergency Mosquito Vector Control Operations. (In press).

Ellis, R. and Brust, R. 1982. Effectiveness of the Emergency Mosquito Vector Control Operations. (In press).

Lillie, L., Wong, F.C., and Drysdale, R.A. 1976. Equine Epizootic of Western Encephalomyelitis in Manitoba - 1975. Can. J. Public Health (Suppl. 1):21-27.

Sekla, L. and Stackiw, W. 1976. Laboratory diagnosis of Western Encephalomyelitis. Can. J. Public Health (Suppl. 1) 67:33-39.

Waters, J. 1976. An Epidemic of Western Encephalomyelitis in Humans - Manitoba, 1975. Can. J. Public Health (Suppl.)67:28-32.

Wong, F.C. and Neufeld, J.L. 1982. Sentinel Flock Monitoring for WEE in Manitoba 1976-1981. (In press).

Table 1

Sentinel Flock
Seroconversion to WEE After Aerial Adulticiding

UNTREATED	EXPOSURE PERIOD	TOTAL DAYS	PERCENT SEROCONVERSION
Traverse Bay	Aug. 1 - Aug. 31	30	0
Oak Hammock	Aug. 5 - Sept. 15	41	15
Arborg	Aug. 1 - Aug. 31	30	50
Glenlea	Aug. 12 - Sept. 14	40	7
TREATED			
U of M	Aug. 14 - Aug. 27	13	14
Morden	Aug. 14 - Aug. 28	14	0
Virden	Aug. 15 - Sept. 9	25	6
Morris	Aug. 14 - Aug. 28	14	0
Steinbach	Aug. 14 - Sept. 4	20	0
Dauphin	Aug. 19 - Sept. 8	20	0
Charleswood	Aug. 12 - Sept. 10	29	60
Portage la Prairie	Aug. 12 - Sept. 14	33	66
Brandon	Aug. 15 - Aug. 26	11	21
Selkirk	Aug. 14 - Aug. 28	14	50

CHAPTER V:

Virology

Arbovirus Isolations and Serology in Manitoba

L. Sekla M.B.B.Ch., Ph.D.
W. Stackiw B.Sc.

Manitoba Health Services Commission
Cadham Provincial Laboratory 750 William Avenue
Winnipeg, Manitoba R3C 3Y1

Abstract

A total of 121 arboviruses were isolated from 4,153 pools of mosquitoes; of these, 113 were Western Equine Encephalitis (WEE) viruses.

Sentinel chicken flocks demonstrated a greater than 20% seroconversion per location and cycle only during the epidemic years, 1975, 1977 and 1981.

A total of 565 clinically suspect horses were tested; of these, 330 were serologically confirmed as recent WEE cases.

A total of 1,304 humans with histories suggestive of WEE infection were tested; 44 were serologically confirmed as recent WEE cases and 24 as presumptive WEE (based on the presence of high static antibody titres). Three cases of St. Louis Encephalitis (SLE) were also diagnosed serologically.

The number of reservoir hosts tested for antibodies to WEE, California Encephalitis (CE) and (SLE) was too small to warrant any definite conclusion. Serosurveys of healthy humans conducted in 1968, 1980 showed that 5 and 8% of the population has been exposed to WEE.

Arboviruses in Canada have been reviewed by McLintock (McLintock and Iversen 1975) and Artsob (Artsob and Spence 1979). Western Equine Encephalitis (WEE) was recognized as the sole mosquito-borne virus causing human epidemics in Canada (in particular in the Prairies) until 1975 when Ontario experienced an outbreak of St. Louis Encephalitis (SLE). Since then, another mosquito-borne arbovirus, California Encephalitis (CE) has been reported from humans in Quebec. Two other mosquito-borne viruses, Hart Park Flanders (HPF) and Cache Valley (CV) have not yet been associated with disease in humans. In Manitoba, WEE is the commonest arbovirus infection, the last major outbreak in humans occurring in 1941 with 79 deaths. During the years 1975 - 1981 all tests for arbovirus isolations and serology were performed at the Cadham Provincial Laboratory (CPL). We propose to review the procedures used and the results obtained.

MATERIALS

A total of 4,153 mosquito pools were processed for arbovirus isolation while 5,458 sentinel chickens and 565 equine bloods were tested serologically; the collection and submission of these samples were described in Chapters II, III, and IV respectively. In addition, bloods from a variety of suspected reservoir hosts were tested for the presence of arbovirus antibodies; included in these studies were 174 garter snakes, 16 ground squirrels, 19 Richardson squirrels, 3 gophers, 11 pigeons, 31 rabbits and 21 turkeys.

A total of 1,304 humans with Central Nervous System (CNS) symptoms were tested serologically. As almost all the serological testing done in the Province of Manitoba is centralized at the Cadham Provincial Laboratory (CPL), it facilitates the selection of those with a symptomatology compatible with the diagnosis of WEE such as pyrexia of unknown origin, encephalitis, meningitis, severe headache, high fever with or without stiff neck, etc. Also in 1981 cerebrospinal fluids were examined for virus and found negative.

In addition to tests on patients, several serosurveys were conducted on a total of 3,490 persons; of these, 3,775 were healthy persons submitting bloods for the compulsory syphylis serology testing, 540 were children and 25 senior citizens (the last two groups with symptoms unrelated to arboviral infections); finally 150 persons were included in the serosurvey as family contacts of proven WEE cases or because of a known occupational risk of exposure to the mosquito vectors.

METHODOLOGY

A. Arbovirus Isolation from Mosquitoes

Mixed mosquito species and sexes were processed in 1975. However, since 1976, females only were sorted by species and location and brought to the laboratory. Pools of live mosquitoes were inactivated by chilling for five minutes in a freezer and were ground immediately with a tissue grinder (Ten Broek) in a pH 7.4 diluent consisting of BME tissue culture media containing 25% Kaolin adsorbed fetal calf serum with 1000 IU of penicillin, 250 µgms of streptomycin, 50 µgms of chlormycetin, 50 µgms mycostatin and 500 µgms gentamycin per ml. The volume of diluent varied from 1 to 10 ml depending on the size of the pools. The suspensions were centrifuged at 4°C for 30 minutes at 1000 x g. Equal volumes of five mosquito pools were combined and used for mouse inoculation; single pools were inoculated into duplicate tissue cultures either VERO, BHK-21, or primary AFGMK, and then stored at -70°C.

Tissue cultures were incubated at 36°C and read daily for cytopathic effects. Cultures that were negative after seven days were frozen and thawed and were then repassed into fresh cultures and incubated for seven days. Those showing a cytopathic effect were identified by neutralization with WEE immune serum (mouse ascitic fluid).

A litter of mice, two or three days of age, was inoculated with the combined pool, 0.02 ml intracranially and 0.05 ml intraperitoneally per mouse. Mice were observed for 14 days; those that became ill 24 hours after inoculation were sacrificed, frozen, thawed and the brain tissue removed with a syringe and needle. A 20% suspension of the brain was made in diluent and tested for sterility. Bacteria-free suspensions were diluted 1 in 10 in diluent and inoculated into a litter of mice. A 20% brain suspension of dead mice was repassed into tissue culture and identified, where possible, by neutralization.

Brain suspension and tissue culture fluid were also sent to the National Arbovirus Centre, Toronto, for confirmation or identification.

B. Arboviral Serology

Four types of serological tests were performed at the CPL: i.e.,

A haemagglutination-inhibition (HI) test was used for screening all human, equine and chicken sera from 1976 - 1981.

A complement fixation test (CF) was used for screening human and equine sera in 1975, but restricted to confirming HI positive samples since then.

A neutralization test (Nt) was performed in some serosurveys and to assess the specificity of the HI test.

In 1981, selected human sera suspected of having recently acquired a WEE infection were tested for the presence of specific IgM antibodies.

In 1976 - 1980, human sera were tested for SLE and CEV, as well as WEE antibodies.

Criteria for the serological diagnosis of a recent arboviral infection during an epidemic included: i.e,

The demonstration of at least a four-fold rise in antibody titres in paired sera tested simultaneously.

The presence of both CF antibodies (>1:8) and HI antibodies (>1:32) in single sera, or in paired sera showing static titres.

The presence of immunoglobulin M (IgM) antibodies specific for WEE.

The serological tests can be detailed as follows:

Hemagglutination - Inhibition Test (HI)

The procedure used at the CPL was a modification of that recommended for Rubella HI testing and has been described by Sekla and Stackiw (Sekla and Stackiw, 1976).

Complement Fixation Test

The method used has been described previously by Sekla and Stackiw (1976) and is essentially that described by the Centre of Disease Control (CDC), Atlanta (Anonym. 1974).

Neutralization Tests

Method used was basically the one described by Lennette, E.H. and Schmidt, N.J. (Lennette and Schmidt 1979). Briefly, sera were inactivated at 56°C for 30 minutes and diluted in BME tissue culture (TC) media containing 1% fetal calf serum. To an equal volume of serum dilution a 50 - 100 TCD_{50} of virus was added; the serum-virus mixture was incubated at 37°C for one hour and inoculated into either primary African Green Monkey Kidney TC or chick embryo fibroblast TC. Cultures were read on the second or third day and the endpoint was read as the highest dilution of serum that competely neutralized the viruses.

In the 1968 serosurvey, sera were screened at two dilutions, 1 in 2, and 1 in 4. Those found to be positive at 1 in 4 were carried out to titre. The strain of virus used was a Manitoba isolate from 1966; the TC used was chick embryo fibroblasts. In 1981, the virus used was the first strain isolated that year from mosquitoes.

Immunoblobulin M Determinations

Immunoglobulin fractions were separated by a sucrose gradient fractionation procedure (Vesikari, T. and Vaheri, A. 1968). Briefly five concentrations of sucrose ranging from 37.5% to 12.0% were prepared; then 0.3 ml of serum was layered on the top fraction and centrifuged at 35,000 RPM (Beckman Model L65, Rotor 50-1) for 18 hours. Eight 0.5 ml fractions were collected and each fraction was diluted serially and tested for antibody for WEE by the HI procedure. Detection of antibody in the fractions previously determined to contain IgM fraction was considered positive.

RESULTS

Arbovirus Isolation

The number of arbovirus isolations from mosquitoes during the years 1975 - 1981 (has been reported in Chapter II) and is shown in Table 1 which indicates the number of mosquito pools tested, the number and type of arbovirus isolations, as well as the percentage of positive pools. A total of 121 arboviruses have beer isolated from mosquitoes: 113 WEE, 4 CV, 2 CE, and 2 HPF. One of the CE isolated from *Aedes* sp was identified as a Jamestown Canyon subtype; the other isolated from *Culiseta inornata* was identified by the National Arbovirus Reference Center as a Snowshoe hare subtype.

Serology

Sentinel chicken flocks results have been discussed in Chapter IV. From 1975 until 1981, HI antibody titres of 1:32 or greater were detected as shown in Table 2; there was a marked difference in seropositivity between WEE epidemic and non-epidemic years. During epidemic years, 16% of all the chickens tested developed antibodies while less than 4% did so in non-epidemic years.

Equine

Of the 565 sick horses tested, 330 or 58.4% were found to have WEE antibodies. Table 3 shows the number of laboratory confirmed equine cases of WEE reported each year, clearly showing epizootics.

Reservoir Hosts

Details of the serological tests are given below (Table 4). The results were negative with the exception of 2 squirrels and 4 turkeys which had WEE antibodies and 1 rabbit with antibodies to the St. Louis encephalitis virus. Overall, 29% of the 31 rabbits showed serological conversion to CE. Of these 9 out of 12 (75%) sentinel rabbits set out in NW Manitoba showed serological conversions by August 8th. None of the 12 rabbits located in Southern Manitoba were positive.

Sick Persons

The number of sick persons tested in each of the years 1975 - 1981 is given in Table 5, as well as the number of confirmed cases and the number of presumptive cases of arboviral infections (defined as sick persons with static titres). During these years, 44 confirmed and 24 presumptive cases of encephalitis were due to an infection with the WEE virus; three cases were due to the SLE virus and none to the CE virus. Two deaths were attributed to WEE in 1981.

Details of the 1981 epidemic, showing the date of onset of clinical symptoms and the dates of collection and results obtained on the first and last bloods tested, as well as those of the first sample reported as positive are given in Table 6. All 25 bloods were found to have neutralizing antibodies at titres ≥1:4.

In these 25 patients, the serodiagnosis was based on a four-fold rise in HI antibodies in 18; on the presence of specific IgM in 5; and on the presence of CF antibodies at titres >1:8 with static HI titres in 2. Antibodies were detected in the first blood in only 14 patients; the diagnosis was confirmed in 4 - 29 days.

Human Serosurveys

The various serosurveys done in Manitoba are summarized in Table 7. In the premarital population, antibodies to WEE were detected in 5.2% and in 8% of those tested in 1968 and 1981 respectively; antibodies to CE were detected in 7.2% of 607 persons tested in 1979; those residing in the NW Manitoba had a higher incidence of 12.5% with 36.6% positive in the 51 or greater age group. Antibodies to SLE virus were detected in 3 of 25 sera tested in 1977.

DISCUSSION

The procedures used for the isolation of WEE virus have allowed the identification of other arboviruses. It is interesting to note that although there is serological evidence that SLE exists in Manitoba, no virus isolation from mosquitoes has been made to date. In contrast, the CE virus has been isolated from mosquitoes; both subtypes identified were thought to be rare in Canada, and isolates were obtained from mosquitoes collected from two neighbouring localities in SE Manitoba. The presence of CE antibodies in 29% of the sentinel rabbits and in 7.2% of the Manitobans tested indicate that the CE virus is widespread.

Arboviral serology can be extremely useful in monitoring infections in horses, chickens, as well as in a variety of reservoir hosts. In particular, sentinel chicken flocks can be a sensitive tool in predictive surveillance programs. Monitoring sick horses is more a diagnostic than a surveillance procedure. However, since epizootics of WEE in horses (defined as more than six cases confirmed per week) often preceed epidemics in humans by 2 - 3 weeks, equine surveillance is a vital warning system; the epizootic started two weeks before the 1975 and 1977 epidemics and three weeks before the 1981 one.

The number of reservoir hosts tested is too small to warrant any definite conclusion. Yet a study of reservoir hosts would be of value in determining the extent of arbovirus infection in wild life. The presence of WEE antibodies in turkeys from the farm of one of the confirmed WEE cases illustrates that residents, as well as sentinel birds, may be used in the surveillance.

The serological tests done on sick persons include tests for CE, SLE, WEE, *Herpes simplex*, as well as a battery of other viruses likely to affect the central nervous system. No case of CE has been diagnosed so far. Nine persons had antibodies to group B arboviruses; six of those had a history of travel to the Caribbean Islands and were diagnosed as Dengue fever, while three had not travelled outside the Continental North America and were classified as SLE. In 1981, three of the confirmed cases of WEE had concurrent infections with Coxsackie B viruses. In the same year, no WEE antibodies were detected in 30 persons with definite encephalitis and/or myelitis; of these two were proven to have *Herpes simplex* encephalitis and one had a Hepatitis B viral infection.

Over the years a variety of serological tests have been used, indicating that no test is entirely satisfactory. However, the HI test is the most suitable one for screening purposes. It is simple, sensitive and reproducible. Two false positive results only were reported in 1981; the serum was lipaemic in one case and was grossly contaminated in the other. HI antibodies to WEE are reported to rise early in the infection and to last almost indefinitely. HI antibodies were detected in 1975 in a Manitoban known to have been infected in the forties. However, the time of appearance of HI antibodies is variable, for example, 11 of the 25 cases confirmed in 1981 had no antibodies in the first blood collected 3 to 12 days after the onset of clinical symptoms; therefore, frequent serial testing is essential. Serological confirmation was obtained 21 to 29 days after the onset of clinical signs in four of the 25 cases diagnosed in 1981.

The Complement-fixation test is of limited value in the serodiagnosis of arboviruses in Manitoba. CF antibodies are slow in developing; to illustrate this point the 1981 experience will be detailed; WEE antibodies were detected in the first sera of only two of the confirmed and one of the presumptive WEE cases; in ten of the confirmed cases, CF antibodies developed 4 - 50 days later; in all the others, (13 confirmed and eight presumptive cases), CF antibodies were not detected up till the last serum submitted. CF antibodies are said to be short-lasting (<6 years), however, early in 1982, six victims of the 1975 epidemic were retested by CF and all six of them were found to be still positive.

The detection of specific IgM antibodies is a valuable tool permitting a serodiagnosis on a single early serum. However, the separation of immunoglobulins by sucrose gradient centrifugation is too complex to be used as a screening procedure. The longevity of the specific IgM antibodies has yet to be determined; they are expected to be short-lived and their presence indicate a recent infection. In 1981, IgM antibodies were detected in eight of the 28 persons tested; however, in retrospect one of the confirmed cases was found to have no IgM antibodies; this person reported having symptoms compatible with a WEE infection earlier on in the summer and a relapse in August.

The Neutralization test is both sensitive and specific, however, it requires culture and biohazard facilities. This test was used for a serosurvey in 1968. In 1981, the Nt test was used to confirm the specificity of the 41 sera found to be positive and the 30 sera found negative by HI. There was a good correlation between the two tests.

The main disadvantage of serodiagnosis is the need for repeated serial testing. For example, single sera only were submitted in 41% of the 581 patients investigation in 1981. Another difficulty arises from subtle differences in the antigenic make-up of strains of viruses. Antibody titres may be lower if the strain of WEE used in the test is different from the current infective one. This hypothesis may explain the low level of antibodies detected in 1981 in both the HI and the CF tests.

Serosurveys were conducted in an attempt to define the extent of arboviral infections in Manitoba. The only study with adequate number and stratification is the 1968 survey (unpublished data - Snell, E. et al 1968) which will be reviewed briefly. In the premarital population tested by Nt, the overall incidence of antibodies was 5.2%, with an equal distribution of antibodies in males and females and in those borne before and after the 1941 WEE epidemic. In three of Manitoba's Census divisions, the percentage of persons with antibodies exceeded 5.2%, two of these census were in the SW part of the Province where 12% and 17% of the persons tested were found to have antibodies; the third census was in Portage la Prairie where 8% had WEE antibodies. In the same 1968 serosurvey, 208 children, age one month to 15 years were tested by Nt, but none had detectable antibodies. As well, 25 residents of an Old Folk's home with a past history suggestive of an encephalitis were tested and WEE antibody found in 28% of them. When a much small number of premaritals were tested by HI in 1981, 8% were found to have antibodies to WEE, despite three outbreaks in the interval between 1968 and 1981. It is worth noting that the same percentage of antibodies (8.4%) was found in family contacts of cases of WEE and in persons occupationally exposed to mosquitoes during the summer of 1981. The serosurveys have indicated a high level of susceptibility in humans; it is interesting to speculate that perhaps only individuals with certain genetic characteristics are affected by WEE.

Since the exact time of an infection cannot easily be defined serologically it has been impossible, so far, to determine the ratio of symptomatic to asymptomatic infection in Manitoba.

CONCLUSIONS AND RECOMMENDATIONS

Arboviral isolation and serology play an important role in predicting outbreaks of WEE with confidence. By restricting the testing to the months of July and August and by concentrating mainly on Cx tarsalis, viruses can be isolated from mosquitoes within a week of collection. Research is needed to develop more rapid laboratory techniques such as, for example, an ELISA (Enzyme-linked immunosorbent assay) testing using monoclonal antibodies to detect IgM antibodies. An analysis of antigenic make-up and finger printing of the isolates is worth attempting.

Acknowledgements

We thank the National Arbovirus Reference Centre and the STEP program for their contribution to this work. Our gratitude goes to the staff of the Environmental Microbiology and Serology sections of the Cadham Provincial Laboratory, to Dr. J. C. Wilt, Director of the CPL and to Marie Myndzak who patiently typed all the manuscripts.

References Cited

Anonym. 1974. U.S. Department of Health, Education and Welfare, Standardized Diagnostic complement fixation method and adaptation to micro test. Public Health Monograph. No. 74, Revised December 1974. 56 pp.

Artsob, H., Spence, L. 1979. Arboviruses in Canada. In Kurstak, E., (Ed.), Art. Trop. Arboviruses. Academic Press, New York, NY 39-65.

Lennette, E.H., Schmidt, N.J. 1979. Diagnostic Procedures for Viral Rickettsial and Chlamydial Infections. 5th Edition. 1138 pp.

McLintock, J., Iversen, J. 1975. Mosquitoes and human diseases in Canada. Can. Ent. 107:694-704.

Sekla, L., Stackiw, W. 1976. The Laboratory Diagnosis of Western Encephalomyelitis. Can. J. Public Health. Suppl. 1 to Vol. 67:33-39.

Snell, E. et al 1968. Antibody level against WEE in man, in different age groups and in different geographical areas in Manitoba. Unpublished data.

Vesikari, T., Vaheri, A. 1968. Rubella: A method for rapid diagnosis of recent infection by demonstration of the IgM antibodies. Br. Med. J. 1:221-223.

TABLE 1

ARBOVIRUS ISOLATIONS FROM MOSQUITOES

	1975	1976	1977	1978	1979	1980	1981	
POOLS TESTED	72	882	1045	581	450	395	728	= 4153
ARBOVIRUS ISOLATED	5	0	73	1	7	0	35	= 121
% POOLS POSITIVE	7	–	7	0.1	1.6	–	4.8	
WEE	5	–	71	1	4	–	32	= 113
HPF	–	–	–		1	–	1	
CVV	–	–	2	–	–	–	2	
CEV	–	–	–	–	2	–	–	

TABLE 2

SENTINEL CHICKEN FLOCKS 1975-1981

	*1975	1976	*1977	1978	1979	1980	*1981
No. positive / No. tested (i.e.: HI ≥ 1:32)	214/820	8/1080	135/830	19/600	6/1041	5/743	63/344
% positive	26	0.98	16.2	3.1	0.5	0.6	18.3

*Epidemic year

TABLE 3

EQUINE CASES OF W.E.E. 1975-1981

	1975	1976	1977	1978	1979	1980	1981
SUBMITTED	261	19	80	5	6	11	183
CONFIRMED	145	10	53	1	0	1	120

TABLE 4

SEROSURVEYS-Manitoba

Reservoirs

	Year	No. tested	Arbovirus	Test screening dilution	Percent positive at screen
Garter snakes	1966	174	WEE	CF 1:4	0
Ground squirrels	1966	16	WEE	CF 1:4	0
Gophers	1966	3	WEE	CF 1:4	0
Richardson squirrels	1977	19	WEE	HI 1:8	10.5%
Rabbits	1977	64	SLE	HI 1:10	6.2%
Pigeons	1977	11	WEE	HI 1:8	0
Rabbits	1979	31	CE	HI 1:10	29%
Turkeys	1981	21	WEE	HI 1:8	19%

TABLE 5

HUMAN ARBOVIRAL INFECTIONS, 1975-1981

	* 1975	1976	* 1977	1978	1979	1980	* 1981
No. PATIENTS TESTED	196	111	256	14	54	87	586
No. CONFIRMED WEE	14		5				25 (2 deaths)
No. PRESUMPTIVE** WEE	9		6				9
OTHER ARBOVIRAL CASES	1 SLE		2 SLE				

* EPIDEMIC YEARS ** STATIC TITRES

TABLE 6
1981 SEROLOGICALLY CONFIRMED CASES OF WEE

Patient	Date onset of symptoms	Date of collection and results										
		First blood				Blood confirmed positive				Last blood tested		
		Date	HI	CF	IgM	Date	HI	CF	IgM	Date	HI	CF
1. EK	13/7	19/7	<8	<4		2nd wk. Aug	128	8	+	1st wk. Sept	256	16
2. BM	1/8	8/8	16	<4		13/8	128	<4		25/8	128	<4
3. PR	28/7	7/8	<8	<4		20/8	128	<4				
4. MP	25/7	Last wk. July	<8	<4		4/8	32	<4		19/8	64	4
5. SA	5/8	15/8	16	<4		25/8	64	4		19/10	256	32
6. MA	20/8 (Died)	24/8	<8	<4		28/8	64	<4		4/9	1024	8
7. HC	23/8	26/8	<8	<4		31/8	64	<4		8/9	128	4
8. BJ	10/8	24/8	32	<4		31/8	128	<4		10/9	64	NSQ
9. CG	13/8	31/8	64	16	–	11/9	128	16		28/9	128	
10. McGT	24/8	1/9	32	<4		3/9	128	<4				
11. YE	14/8	18/8	<8	<4		2/9	32	<4		1/10	64	16
12. SJ	28/8	4/9	16	<4		18/9	64	<4		13/10	16	<4
13. CL	5/8 (Died)	12/8	64	<4	+	20/8	64	<4				
14. MD	27/8	1/9	<8	<4		6/9	32	<4		15/9	64	<4
15. KS	4/9	5/9	32	4		11/9	64	8				
16. KW	19/8	24/8	<8	<4		10/9	512	<4				
17. WC	17/8	21/8	8	<4	+					28/8	16	<4
18. WR	6/8	10/8	16	<4	+					16/9	64	16
19. BE	15/8	27/8	<8	<4						11/9	64	<4
20. VM	3/9	21/9	16	<4	+					2/11	128	<4
21. IP	27/8	31/8	<8	<4		14/9	16	<4	+	8/10	64	<4
22. FJ	3/9	8/9	<8	<4		10/9	16	<4		30/9	512	32
23. BP	1/10	9/10	16	<4	+					27/10	16	<4
24. McKD	14/9	13/10	16	<4	+					11/11	32	<4
25. GC	7/7	30/7	32	<4	+					14/9	32	<4

HI: Haemagglutination-inhibition test
CF: Complement-fixation test
NSQ: Not sufficient quantity

TABLE 7

SEROSURVEYS–Manitoba

Humans

Year	No. tested	Arbovirus	Test α screening dilution	Percent positive at screen
1968	1863	WEE	Nt 1:2	5.2%
1975	492	WEE	HI 1:8	0.8%
1977	25	SLE	HI 1:10	12%
1979	607	CE	HI 1:10	7.2%
1981	a) 100	WEE	HI 1:8	8 %
	b) 150	WEE	HI 1:8 Nt 1:4	8.6%

CHAPTER VI:

Clinical and Epidemiological Data

Clinical Study of Western Equine Encephalitis 1981

B. Friesen, M.D.
J.A. Eadie, M.B.Ch.B., D.P.H.

Preventive Medical Services
831 Portage Avenue
Winnipeg, Manitoba
R3G 0N6

Abstract

All cases of Western Equine Encephalitis (WEE) reported during an epidemic in Manitoba in 1981 were studied and followed up. An analysis of their illnesses showed that most symptoms of WEE are non-specific. The clinical picture is unchanged from past studies. There is significant morbidity and mortality associated with WEE infection.

The objectives of this case study were to provide detailed information on the clinical course and outcome of persons who were reported as having WEE. Similar information has been reported previously (Adamson and Dubo 1942). They reported on an epidemic of 509 suspect human cases, however their diagnostic methodology was probably not very accurate. No epidemic of similar size has been reported since. The 1981 epidemic with 25 reported cases was the largest in Manitoba since 1941 and presented an opportunity to document the clinical course. A report in 1976 had provided information on 14 cases (Waters 1976). One hypothesis for the reduction in the numbers of human cases over the years since 1941 was a change in the virulence of virus resulting in a milder illness. A comparison of the 1981 cases with past cases will address this issue.

MATERIALS AND METHODS

The 25 cases that are presented in this study are those people who were reported to Preventive Medical Services as having WEE. The laboratory diagnosis of these cases is reported elsewhere (Sekla and Stackiw 1982). The cases were initially identified by their attenting physician as suspected WEE cases or detected as part of a surveillance program run by the Cadham Provincial Laboratory. The data were obtained from attending physician records, hospital records, patient interviews and family interviews. The interviews took place between 1 week to 6 months after illness, the majority 4 - 5 weeks after onset.

RESULTS

WEE was rarely mentioned as one of the differential diagnoses on either the laboratory requisitions or on the attending physicians' records. Cases tended to be persons who were very active outdoors, often working outside. They continued to work after the onset of their illness until they were forced to stop. The recovery from WEE was slow. The difficulty of making the diagnosis is illustrated by the following case history which is representative of other patients' illnesses.

CASE HISTORY

One of the confirmed cases in 1981 was a 26 year old farmer. In addition to farming he was an active competitor on the rural Manitoba rodeo circuit. As a result he had travelled widely in southern Manitoba attending various 1 or 2 day rodeos. His potential exposure to infective mosquitoes while farming was great, especially since the tractor and the swather he operated were not enclosed. He first began feeling unwell when he developed a headache, which was constant and originated in the back of his neck. The pain was increased with neck movement. He slept with a pillow under his shoulder to extend his neck and relieve the discomfort. He continued to work long hours and on day 3 of his illness he entered a rodeo in which he won one of the events. By that evening, his headache was more severe. He became nauseated and began to vomit. On the following day he was lethargic and slept the whole day. He was seen by his physician the next day, day 5. Fever was the solitary sign. It was felt in view of the gastrointestinal symptoms that he had a flu-like illness. Antipyretics and analgesics were prescribed with instructions to return, if there was no improvement. On day 6, his wife returned from work to find her husband confused. He did not recognize her and he had a high fever with rigors. She brought him to the emergency department, where, in addition to the fever and confusion, he had a fine tremor of both hands. He was hospitalized. A lumbar puncture was attempted but was unsuccessful. On day 7, because of persistant fever and confusion, he was transferred to a tertiary care hospital. Upon admission, he was pyrexic. His speech was slurred and incoherent. He was confused and incontinent of urine. Physical examination revealed a temperature of 38.8°C but was otherwise negative. His neck was supple. There were no neurological signs. A lumbar puncture was done. The pressure was normal. There were 35×10^6 cells/L (35/mm3) - all lymphocytes. There were no organisms on gram stain and bacterial culture was negative. The sugar was 0.58 g/L (58 mg/dl) and protein was 0.64 g/L (64 mg/dl). His peripheral white blood cell count was 9.5×10^9 /L (9500 per cu. mm.) with 62% polymorph and 34% lumphocytes. A brain scan was done on day 8, and showed no focal or diffuse activity and was interpreted as normal.

In hospital, he gradually improved. By day 10, he was able to walk, although he was unsteady. He became afebrile on day 11. On day 12 he was fully oriented, with no further episodes of confusion. At discharge, 14 days after the onset of his illness, his hands were still very tremulous and he had generalized weakness. The only medication he received in hospital was Largactil prior to transfer between hospitals. He did not receive any antibiotics.

Antibodies to WEE viurs were not detected in a serum specimen taken on day 9 of his illness while a specimen on day 21 had a titre of 1/512 by hemagglutination-inhibition. When he was interviewed 2 months after his discharge, he still was not completely recovered. He complained of decreased endurance. He still had a tremor in his right hand which affected his writing and he complained of forgetfulness. His wife also mentioned that he tended to be more irritable than before.

SYMPTOMS AND SIGNS AT ONSET

The symptoms and signs manifested by the 25 cases at the time they were first seen by a physician are shown (Table 1). Fever and headaches were the most common. They were present in 72% and 60% of the cases. Gastrointestinal symptoms and drowsiness occurred in about a third of the cases. Confusion was intermittent at this stage of the illness. It may not have been present when the patient was seen but it was possible to elicit a history of it in over a quarter of the cases. Nuchal pain and rigidity were very rare, being found in only 8% of the patients when they were first seen. Generalized weakness and ataxia were the most common signs at the initial physical examination. WEE presented more as a non-specific, flu-like illness than as a CNS infection except that respiratory symptoms were absent.

SYMPTOMS AND SIGNS DURING ILLNESS

The frequency of symptoms that occurred over the first 3 weeks of the illness are shown (Table 2). Drowsiness was present in 88% of the cases and lasted from 2 to 21 days. The average time was 6 days. Headaches occurred in 72% of the people, with median duration of 6.5 days, but some still complained of headaches after 3 weeks. Fever occurred at some point in all but one case, with duration ranging from 4 to 14 days, with a median of 6 days.

Sixty-four percent of the patients were confused at some point in their illness. It tended to be of short duration. The median was 3.5 days. Irritability was common and was persistent. Coma occured in 4 patients. The median duration was 6 days, although one patient was still semi-comatose when he died 4 months later. Two cases had seizures. They were both over 70. The seizures were generalized and occurred on the 2nd and 5th days of illness. Weakness both generalized and localized occurred, generalized being the commonest. Two men had weakness of one of their upper extremities. Another 2 had weakness of their lower extremities including one man who had a transverse myelitis of the thoracic spine due to WEE.

LATE SYMPTOMS

Most people were interviewed at 2 - 3 months after the onset of the illness. They were not examined. Their complaints or symptoms at that time are shown (Table 3). Over half the cases had residual symptoms. Cases classified as "mild" averaged 31 years of age and had fully recovered or had only one symptom. This was usually fatigue. "Severe" cases averaged 53 years of age and most had 2 or more symptoms. Fatigue was the most common residual symptom. One man, describing its effect on him, said, "work took twice as long and was twice as hard to do" after he was sick. The fatigue gradually disappeared although early return to work, within 2 weeks after onset of the illness, appeared to prolong the fatigue. Mood change was probably the most severe residual symptom. This manifested itself as depression or irritability and combine with disorders of long term memory had the most impact on the patient and his family. All the residual symptoms seemed to be still improving at the time cases were interviewed, with the possible exception of the memory disorders.

Eighteen (72%) of the cases were hospitalized. The length of their hospitalization ranged from 3 days to 4 months. The median stay was 9 days. Fifteen (60%) had one or more lumbar punctures done - 3 - 12 days after the onset of the illness. The median was 6 days. This was somewhat later than would be expected as the median time after onset for seeing a physician was 2.5 days, while median time from onset until admission to hospital was 3 days. The results of the lumbar punctures are shown (Table 4).

INVESTIGATIONS.

Spinal pressure was recorded infrequently and in most cases where it was reported it is uncertain whether it was initial or final. In one instance the initial was 320 cm H_2O and it fell to 180 cm H_2O at completion. There was elevation in some cases. Examination of the Cerebro Spinal Fluid (CSF) usually demonstrated a pleocytosis with a lymphocytic predominance. Some CSF specimens showed polymorphs mainly. The protein was either normal or slightly to moderately elevated. Nine of the patients had Electro Encephalograms (EEG) done. All were abnormal. The electrical distrubances were diffuse in all cases. It was reported as non-specific slow wave activity in 4. Theta and delta wave activity were reported in one instance each. The other 5 EEG reports did not give detail on the type of activity. Nine patients had Computerized Axial Tomography (CAT) scans. In 5 instances they included infusion studies. Cerebral atrophy was present in 2 patients. One was 55 years old, while the other was 70 years old. All other CAT scans were normal. Brain scans were done in 6 patients and all were normal.

DRUG THERAPY

Eleven patients received antibiotics. The duration of antibiotics was 1 - 8 days, the median was 4 days. Chloramphenicol and ampicillin were the most commonly prescribed medications. Two patients received adenine arabinoside (Ara A) on suspicion of herpes encephalitis. They were both comatose. One of the patients presented with fever, headache, malaise and left hemi-paresis. She then developed an expressive aphasia before becoming comatose. She had a normal CAT scan. A carotid angiogram was done which showed normal cerebral vasculature but it demonstrated a dissecting thoracic aneuryrm with a left hemathorax. A brain biopsy was performed. The histology showed perivascular leukocyte infiltration. There was no virus demonstrated by electron microscopy. Animal inoculation and tissue cultures of biopsy material did not isolate any viral agents. She received a 5 day course of Ara A and by day 13 of her illness she was no longer comatose. Her level of consciousness was still fluctuating but she was able to talk again. She died suddenly 2 days later. The autopsy showed gastric aspiration, a dissecting aneurysm of the aorta with leakage, a left hemathorax, adrenal gland hemorrhage. The other patient was a male in his mid-fifties. He had been comatose since the onset of his illness. His level of consciousness seemed to be improving slowly. He had repeated aspirationpneumonias which slowed or reversed his improving level of consciousness. He died 4 months later after the onset of his infection while recovering from surgery to correct tracheal stenosis which had occurred as a result of intubation and mechanical ventilation in the acute phase of his illness. No autopsy was done. The death certificate listed the cause of death as respiratory arrest.

DISCUSSION

The initial symptoms in patient with WEE are non-specific, making the diagnosis difficult. A study of patients from WEE epidemics in California (Kokernot et al 1953) reported 20 different initial diagnoses in people subsequently demonstrated to have WEE. The type and frequency of symptoms reported by patients in the 1981 Manitoba epidemic of WEE are similar to those reported during the 1941 Manitoba epidemic (Adamson and Dubo 1942) and the California epidemics (Kokernot et al 1953). However, there are some differences between them. Adamson and Dubo reported the severe headaches were generally frontal, while in the California and Manitoba studies mentioned above occipital headaches were more common. WEE infections were as severe in 1981 as they were in 1941 (Adamson and Dubo 1942). In 1981, four (16%) patients were comatose, 2 (8%) had seizures and 4 (16%) had a paresis which was severe in 3 of the 4 patients. One patient had a transverse myelitis and paresis of both legs and was unable to walk for 10 days; however by 5 weeks he apparently had made a complete recovery. Another patient still had some weakness in his arm interferring with his work, one year after infection. The third one likely had a combination of paresis and disuse atrophy which required him to use a walker for 6 months. During the 1941 epidemic, the same percentage of cases were severely affected; however, the actual number of severely affected cases was greater than in 1981, simply because of the larger number of cases infected in 1941. The 2 deaths among the in

1981 reported cases of WEE give a case fatality rate of 8%. Fulton reported a case fatality rate of 9.6% for cases of WEE in Saskatchewan with serological confirmation (Fulton and Burton 1953). Since the means of identifying and confirming cases have varied, comparison of rates between different regions must be interpreted cautiously. Those who died where among the older people affected. More severe illness was seen in the older age groups. Only 1 infant was affected. He recovered with no sequelae, even though he was only 3½ months at the time of infection. Previous epidemics have shown high attack rates for infants under 1 year with severe sequelae for those under 3 months (Medovy 1943).

The fact that the 2 people who received Ara A were the ones who subsequently died raises some concern about its use in patients with WEE. The 2 patients were extremely ill at the time it was decided to give them the medication. They both had difficulty with fluid overloading which is associated with the large fluid volume required to administer the drug. The overloading was not desirable in patients who may already have increased intracranial pressure due to their primary disease. There was 1 case report in the literature where Vira A was administered to a person with WEE with no problems. This was a 17 year old boy who was diagnosed initially as having Herpes Encephalitis. He received a full course of Vira A and made a complete recovery with no sequelae. WEE virus was subsequently cultured from his brain biopsy (Bia et al 1980). In view of the outcome of the two treated cases in 1981, it is important that physicians weigh the risks of drug toxicity against the dangers of not treating a Herpes virus Encephalitis, in deciding whether or not to administer the drug. WEE can mimic Herpes Encephalitis in terms of its clinical presentation, initially with localized findings. This was seen with the lady who died. Testing for the presence of IgM antibodies to WEE virus may be useful in differentiating between Herpes and WEE. CAT scans or brain scans did not provide any positive evidence of WEE infection but may be useful in the diagnosis of Herpes encephalitis. The EEG's confirmed the encephalitis but did not help in the differentiation.

WEE is a severe infection. In this epidemic it was associated with 2 deaths and caused neurological sequelae which were still present at 8 months in 1 individual. Even individuals who apparently recovered completely may have subtle physchological changes which can cause impairment. There is no evidence from the description of the clinical illnesses to support the hypothesis that the virulence of the WEE virus has changed.

Acknowledgements

We would like to thank all the attending physicians for their cooperation in providing information. We would also like to thank Ms. Nila MacFarlane of the Manitoba Central Nervous System Study Group for her help in reviewing medical records. Most of all we would like to recognize

the cooperation of the patients and their families in providing time to be interviewed.

References Cited

Adamson, V.S. and Dubo , S. 1942. Clinical findings in encephalitis (Western Equine). Can. J. Public Health. 33:288-300.

Bia, F. J. *et al* 1980. Western Equine Encephalitis mimicking *Herpes simplex* encephalitis. J.A.M.A. 244, 367-369.

Finley, K.H. et al 1955. Western Equine and St. Louis Encephalitis, Preliminary report of a clinical follow-up study in California. Neurology. 5:223-235.

Fulton, S and Burton, A.M. 1953. After effects of Western Equine Encephalomyelitis infection in Manitoba. Can. Med. Assoc. J. 69:268-272.

Kokernot, R.H., Shinefiled, H.R. and Longshore, W.A. Jr. 1953. The 1952 outbreak of encephalitis in California, differential diagnosis. California Med. 73-78.

Medovy, H. 1943. Western Equine Encephalomyelitis in infants. J. Pediat. 22:308-318.

Menzies, D. W. et al 1968. Western Equine Encephalitis in Saskatchewan - Follow-up of 114 cases from the 1963 and 1965 epidemics. Epidemiological Bulletin (Canada). 12:27-30.

Sekla, L. and Stackiw, W. 1982. Arbovirus Isolations and Serology in Manitoba. (In press).

Waters, J.R. 1976. An epidemic of Western Encephalomyelitis in Humans - Manitoba. Can. J. Public Health (Suppl. 1) 67:28-32.

Table 1

Symptoms and Signs at the Time First Seen by Physician

Symptoms	No. of Patients		Signs	No. of Patients	
Fever	18	72%	Ataxia	6	24%
Headache	15	60%	Disorientation	4	16%
Vomiting	9	36%	Nuchal Rigidity	2	8%
Drowsiness	9	36%	Tremor	2	8%
Anorexia	8	32%	Photophobia	1	4%
Nausea	7	28%			
Chills	7	28%			
Confusion (History)	7	28%			
Weakness	6	24%			
Nuchal Pain	2	8%			

Table 2

Symptoms and Signs Occurring Within the First Three Weeks After Onset

Symptoms	No. of Patients		Range of Duration	Median
Drowsiness	22	88%	4 - 14 days	6
Headache	18	72%	2 - 21 days	6
Chills	17	68%	4 - 21 days	6.5
Anorexia	15	60%		
Irritability	7	28%		
Signs	No. of Patients		Range of Duration	Median
Fever	24	96%	1 - 20 days	3.5
Disorientation (Confusion)	16	64%		
Ataxia	15	60%		
Weakness	14	56%		
Nuchal Rigidity	11	44%		
Tremor	6	24%		
Photophobia	6	24%		
Slow Speech	5	20%		
Coma	4	16%		
Spasticity	3	12%		
Seizures	2	8%		

Table 3

Residual Symptoms at Time of Interview

Symptoms	No. of People Affected	
Easily fatigued	13	52%
Mood change	6	24%
Fine finger control and writing	4	16%
Tremor	4	16%
Difficulty walking	4	16%
Memory lapses	4	16%
Inability to concentrate	3	12%
Headaches	2	8%
Drowsiness	2	8%
Insomnia	2	8%
Hearing Disorder	2	8%

Table 4

Examination Results

Cerebrospinal Fluid	Median	Range
Pressure cm H_2O	185	100 - 320
White Blood Cell 10^6/L (cu. mm.)	46	
Polymorphs	36%	0 - 84%
Lymphocytes	64%	16 - 100%
Sugar mmol/L (mg/dl)	3.5 (61.5)	2.75 - 4.0 (50 - 72)
Protein g/L (mg/dl)	0.54 (54)	0.18 - 0.97 (18 - 97)

Neuropsychological Sequelae After Western Equine Encephalitis Virus Infection

G.A. Hawryluk, BA (Hons), MA, PhD
Section of Behavioural Science
Department of Psychiatry
Section of Rehabilitation Medicine
Department of Medicine
University of Manitoba
770 Bannatyne Avenue
Winnipeg, Manitoba, R3E OW3

G.W. Hammond, BSc, MD, FRCP(C)
Departments of Medical Microbiology and
Medicine, University of Manitoba, and
the Cadham Provincial Laboratory
730 William Avenue
Winnipeg, Manitoba, R3E OW3

B. Friesen, MD
Northern Medical Unit
University of Manitoba
61 Emily Street
Winnipeg, Manitoba

Abstract

Nine patients (8 adults) underwent extensive neuropsychological testing between 6-12 months post recovery of Western Equine Encephalitis virus (WEE) infection. Definite but mild abnormalities were detected in most patients, paticularly in memory and personality functions. These abnormalities appeared to be most marked for individuals with the greatest disability during the acute illness. Based on this assessment of a small number of patients, subtle neuropsychological sequelae of undetermined clinical significance may persist past the early recovery period, even with WEE virus infections which result in only mild to moderate encephalitis during the acute illness.

This report provides a detailed analysis of neuropsychological function of individuals following Western Equine encephalitis (WEE) virus infection. The most severe sequelae of WEE virus infections have been observed to occur in young infants or elderly individuals. Less obvious but possibly important complications of WEE virus infections require more detailed analysis, as can be provided by a battery of standardized, objective neuropsychological tests. The following report is the result of detailed neuropsychological assessment of 8 adults and 1 child infected during the 1981 epidemic in Manitoba, Canada.

MATERIALS AND METHODS

The clinical features of the 9 individuals during their acute illness are documented below (Table 1). A review with a neurologist of the case records of the 9 patients led to the following classification of the clinical manifestations of WEE virus infection: i.e., (A) Confirmed encephalitis - fever, with signs of brain parenchymal inflammation, including an altered level of consciousness with objective neurological signs plus cerebrospinal fluid pleocytosis and an abnormal electroencephalogram (EEG); (B) Probably encephalitis - similar clinical findings but no laboratory confirmation (CSF examination and EEG not performed) and (C) Encephalitis - doubtful - no objective neurological abnormalities and no EEG or CSF examination.

In the series of 25 individuals with WEE infections, 6 patients could not be tested by these procedures - 2 patients died, 1 returned to Australia, 1 was an infant, 1 did not speak English, and 1 patient refused testing. The 9 individuals of this report were selected from the remaining patients on the basis of their willingness to participate in the study and their ability to visit Winnipeg, the location of the testing centre. All tests were administered at one session between 6-12 months following the onset of illness by one of two experienced psychometrists.

The neuropsychological functioning of these patients was assessed by the Halstead-Reitan neuropsychological battery, a very comprehensive battery of tests designed to assess all aspects of brain functioning (Reitan, 1974). This battery, which takes approximately 5-7 hours per patient to administer, assesses sensorimotor and perceptual functioning, attention, concentration and memory, verbal skills, visuospatial skills, novel problem solving and abstraction abilities, as well as overall intelligence. The data on these subjects is summarized in Tables 2-4. Scores falling in the brain damage range (compared to well established normative data) are indicated by an asterisk. In addition to the numerical analyses, each set of test data was clinically interpreted by a neuropsychologist, taking into consideration demographic and educational variables known to be related to neuropsychological test performance (Parsons and Prigatano, 1978). These results are summarized in Table 5.

In addition to neuropsychological test performance, each patient was administered the Minnesota Multiphasic Personality Inventory (MMPI) to assess possible emotional sequelae to the disease.

Follow up interviews were conducted with close family members and each patient's physician to determine their assessment of the extent of recovery at 6 to 8 months post acute illness.

RESULTS

From Table 1, 3 patients had confirmed encephalitis and 5 patients had probably encephalitis at the time of the WEE virus infection. The other patient (GC) had an acute febrile illness with exacerbation of his chronic bronchitis, and although drowsiness and weakness occurred, neurological assessment was inadequate at the time of the acute illness.

As indicated by Tables 2 through 4, considerable variability was noted in these patients' neuropsychological functioning. Summary data is provided in Table 5. Because of the small sample size, statistical analyses were not undertaken, but in all patients, some degree of impairment in neuropsychological functioning was evident. It must be stressed, however, that because of the small sample size these results should be considered with caution.

The results reported are a portion of the total neuropsychological data collected and reflect the salient areas of impairment. Complete data on all subjects are available from the authors.

DISCUSSION

The extent and nature of neurological damage during the acute infection with WEE virus is difficult to fully assess for most patients. However, by clinical assessment, similar to criteria used for St. Louis Encephalitis infections (Brinker and Monath, 1980), we believe that at least 8 of the 9 patients had symptoms and signs compatible with encephalitis, and EEG results were abnormal in all 5 patients on whom the test was performed.

Of the 8 adult patients assessed, all showed indications of impairment in attention and concentration or memory, although the latter appeared more pronounced. In addition, 6 of the 9 patients displayed some disturbance in sensorimotor or perceptual functioning in the tactile modality. Furthermore, higher order problem solving abilities appeared to have been affected in 6 of the 9 patients. In the adult patients, verbal abilities appeared generally unaffected with the exception of one individual who experienced some difficulties in this area, but this may have been related to below average intellectual abilities prior to onset of illness. However, in the child, (TM), marked difficulties were evident in verbal areas which were apparently not in evidence prior to the onset of illness. In this particular case, it is expected that significant academic difficulties may result. In all patients, visuospatial abilities were unaffected.

In 6 of the 9 patients, some degree of psychological difficulty was present. This was characterized primarily by depressive symptomatology, either reflected in the MMPI results or based upon a clinical interview.

Overall, with the exception of the child case (TM) and one adult (RP) it is expected that the degree of impairment in neuropsychological functioning would not exert a profound effect on day to day capabilities. One could speculate that the neuropsychological deficits detected by the tests that were employed may have little clinical significance for some individuals, depending upon the social and intellectual demands required of these individuals in society. However, interviews with close family members confirmed objective changes in memory in 2 individuals (RP and DM, male) and personality changes of irritability, depression and reduced drive in 1 of these (DM, male). The remainder of patients were considered to have made a complete recovery and were functioning similar to their pre-illness levels.

Few previous reports have carefully evaluated possible neuropsychological sequelae in adults following Western Equine Encephalitis virus infection. The criteria of sequelae have been inconsistent between reports, (Herzon et al., 1957) and the early studies of the sequelae following WEE infections included as "major sequelae" such gross abnormalities as paralysis and mental deterioration with retardation. That summary of 9 published studies of sequelae following WEE to 1956 showed that an overall rate of major sequelae was 13.5% (86 of 636 patients). The incidence of sequelae was determined by inclusion of infants as well as adults, and observations were made at between 1 month to 10 years after infection. Herzon's report included observations that personality changes had been listed as prominent residual sequelae but these did not persist beyond 1 to 2 years in the majority of cases. However, he noted that 10 cases of Parkinsonism had occurred in 86 adults collected from 3 studies, and when infants were eliminated from these series, up to 30% of older individuals had a delayed onset of Parkinson-like symptoms 2-3 years after their infection. Fulton and Burton (1953), and Palmer and Finley (1955) had noted a progressive deterioration in a few adults who developed delayed sequelae 2-3 years after WEE infections. The most recent local experience with WEE infections in Manitoba in 1975 documented 1 of 4 adults who had a change in personality and intellectual abilities (Waters, 1976).

The most extensive neuropsychological assessments performed prior to our present study are those of Mulder, Parrott and Thaler (1952). Twelve adults between the ages of 17-58 years were tested shortly after discharge from hospital and 6 months later. At the time of the repeat testing, a psychiatric interview, neurological examination and discussion with the relatives were also carried out. In Mulder's series, 7 of the patients had presented with or progressed to coma during their acute illness. He observed that the early symptoms during the acute illness improved the most and were not significant at follow-up. These symptoms included disorientation, sleep disturbance, hyperreflexia, tremor and loss of motor coordination. The sequelae observed during convalescence did not improve and even progressed in some individuals by the time of the subsequnet visit. Impairment of intellectual functioning was observed at both assessments in 6 individuals who had developed coma in the acute phase of their illness and results did not change in the time interval of the two tests. Personality changes were an important sequelae in this group as well. Of note, the immediate family of infected individuals observed personality and intellectual deficiencies of which the patient was often not aware. In summary, Mulder observed neuropsychological sequelae of a substantial nature in 9 of 12 adults, which correlated to the presence of severe disease, such as coma, with the initial illness. Those with the more mild initial clinical presentation were often able to return to their former level of functioning.

The largest series of observations of sequelae in adults was by Finley et al. (1955). Of 447 confirmed cases over a four year period, 217 were infections of adults. The time interval for assessment of sequelae was 3 months to 4 years after infection. No attempt was made to quantify the extent of disability following WEE infections and no detailed objective neuropsychological studies were performed. It was estimated that not more than 5% of the adults had sequelae of sufficient severity to be of any significance to these patients.

In the most recent detailed assessment of sequelae following WEE infections, (Earnest et al., 1971), detailed evaluations, including neuropsychological tests were performed on 35 individuals, 2-7 years after infection with Western Equine Encephalitis virus. Only 3 adults were assessed by these methods, and abnormalities of these individuals were thought to exist premorbidly rather than being attributable to their WEE infections.

Pathological studies of the few reported cases of autopsy examinations following WEE infections have demonstrated focal areas of tissue rarefaction necrosis, many of which contained inflammatory cells (Smadel *et al*., 1958). A low grade focal cerebritis may be found in some areas of the brain of these acute cases. In chronic WEE infections, severe cystic degeneration and thickening of blood vessels has been noted. One report has documented inflammatory cells at autopsy several years after infection, suggesting the possibility of chronic inflammation following WEE infections (Herzon *et al*., 1957).

Neuropsychological tests of patients, post WEE, require further evaluation in additional patients, together with longitudinal study after recovery from the acute illness. These tests may also be useful to assist the detection and management of sequelae.

Acknowledgements

This study was supported in part by a grant from NHRDP and through the assistance of Dr. J. Eadie, of the Preventive Medical Services Branch of the Government of Manitoba. The technical assistance of Ms. P. Howard and Ms. L. McRae is greatly appreciated.

References Cited

Brinker, K.R. and T.P. Monath, 1980. Chapter 11. The Acute Disease, by T.P. Monath (Ed.). St. Louis Encephalitis. American Public Health Association, 1015 15th Street NW, Washington, D.C. 503-

Earnest, M.P. *et al*., 1971. Neurologic, intellectual and psychological sequelae following western encephalitis. Neurology 21:969-974.

Fulton, J.S. and A.M. Burton, 1953. After effects of Western Equine Encephalomyelitis Infection in Man. Can. Med. Ass. J. 69:268-272.

Herzon, H. *et al*., 1957. Sequelae of Western Equine and other Arthropod-Borne Encephalitides. Neurology (Minneap.) 7:535-547.

Mulder, D.W. et al., 1952. Sequelae of Western Equine Encephalitis. Neurology (Minneap.) 1:318-327.

Palmer, R.J. and K.H. Finley, 1956. Sequelae of Encephalitis. California Medicine 84:98-100.

Parsons, O.A. and G.P. Prigatano, 1978. Methodological considerations in clinical neuropsychological research. J. Consult. & Clin. Psychol. 46:608-619.

Reitan, R.M. and L.A. Davison, 1974. Clinical neuropsychology: Current status and applications. Wiley, New York.

Smadel, J.E. et al., 1958. Sequelae of the Arthropod-Borne Encephalitides. Neurology (Minneap.) 8:873-896.

Waters, J.R. 1976. An Epidemic of Western Encephalomyelitis in Humans - Manitoba, 1975. Can. J. Public Health 67: (Suppl.) 28-32.

Table 1. Features of the illness in patients with WEE infections evaluated by followup neuropsychological tests.

Patient	Sex	Age	Fever	Disorientation	Confusion	Drowsiness	Motor Disability[1]	Hospitalization
KE[2]	M	16	+(6)[3]	+(5)	+(?)	+(3)	+	+(9)
JF[2]	M	33	+(8)	+(8)	+(8)	+(?)	+	+(10)
PM	M	51	+(9)	-	-	+(?)	+	+(7)
GC	M	37	+(14)	-	-	+(2)	+	+(6)
RP	M	55	+(9)	+(2)	+(2)	+(21)	+	-
TM	M	9	+(?)	-	-	+(10)	-	-
DM	F	47	+(?)	-	-	+(?)	+	-
DM[2]	M	29	+(5)	+(?)	+(?)	+(7)	-	+(9)
JB	M	58	-	+(2)	+	+(20)	+	+(2)

[1] Ataxia plus one or more signs of tremor, weakness or spasticity.

[2] EEG test was abnormal.

[3] Duration in number of days.

Table 2. Higher problem solving abilities.

	Summary Indices			Wechsler Adult Intelligence Scale		
Patient	Halstead Impairment Index[1]	Average Impairment Rating[2]	Age Norms[3]	Verbal IQ	Performance IQ	Full Scale IQ
KE	.4	1.16	2	86	105	94
JF	.3	1.33	2	79	75	76
PM	.7*	2.00*	3	84	89	86
GC	.1	0.58	0	121	116	122
RP	.9*	2.00*	2	87	90	87
TM	--	--	--	92	107	100
DM	.6*	1.66	3	93	89	91
DM	0	0.50	0	122	114	122
JB	.4	1.17	1	92	105	97

[1] Halstead Impairment Index ranging from 0-1.0 with scores .5 and above falling within brain-damaged range.

[2] Average Impairment Rating based on Russell, Neuringer, and Goldstein (1978): 0 = above average, 1 = average, 2 = mild impairment, 3 = moderate impairment, 4 and 5 = severe impairment.

[3] Age Norms based on local age norms and reflecting number of scores out of 10 in brain-damaged range.

* Score in the brain-damaged range.

Table 3. Wechsler Memory Scale[1]

Patient	Wechsler Memory Quotient	Semantic Immediate Recall	Memory Delayed Recall	Figural Immediate Recall	Memory Delayed Recall
KE	101	21*	17*	9*	9*
JF	85	3*	3*	11	7*
PM	97	11*	9*	10	6*
GC	138	24	18*	14	14
RP	90	15*	9*	1*	0*
TM	--	--	--	--	--
DM	106	19*	15*	6*	4*
DM	134	34	21	13	13
JB	100	25	20	3*	3*

[1] Impairment assessed according to Russell, Neuringer, and Goldstein (1978) norms.

* Score in the brain-damaged range.

Table 4. Sensory Perceptual and Motor Examination

Patient	Agnosia[1]		Fingertip Number Writing[2]		Memory Stereognosis[3]		Strength of Grip[4]		Tapping[5]	
	R	L	R	L	R	L			R	L
KE	1	0	1	2	10	10	177	170	49	37*
JF	0	0	2	2	17	16	183	175	44	38*
PM	0	0	0	0	10*	16	115	148	47	44
GC	0	0	0	0	15*	9	165	155	52	48
RP	0	4	0	1	9	9	15	21	37	35*
TM	0	3	6	7	11	9	10	8	33	36
DM	0	0	1	0	9	9	23	20	43	36*
DM	0	0	1	1	8	8	54	49	59	55
JB	1	0	2	2	15	14	59	51	48	51

[1] Finger Agnosia errors right and left.
[2] Fingertip Number Writing errors right and left.
[3] Stereognosis performacne times right and left.
[4] Strength of Grip right and left in kilograms.
[5] Number of finger taps per 15-second interval right and left.
* Score in the brain-damaged range.

Table 5
Clinical Interpretation of Neuropsychological Data

							Summary of Impairment	
Patient	Sensori-motor & Perceptual	Attention Concentration Memory	Verbal	Visuo-spatial	Higher Problem Solving	Emotional	Degree	Lateralization
K. E.		+			+		very mild	left
J. F.	+	+	+			+	very mild	left
P. M	+	+			+	+	mild	bilateral, R > L
G. C.	+	+				+	very mild	left
R. P.	+	+			+	+	mild to moderate	right
T. M.	+	+	+		+		mild	bilateral, L > R
D. M. (f)	+	+			+	+	mild	right
D. M. (m)		?				+	age appropriate	---
J. B.		+			+		mild	right

Table 6: Reproduced
From Sekla and Stackiw p. 115
for Easier Identification of WEE Cases

TABLE 6
1981 SEROLOGICALLY CONFIRMED CASES OF WEE

Patient	Date onset of symptoms	Date of collection and results										
		First blood				Blood confirmed positive				Last blood tested		
		Date	HI	CF	IgM	Date	HI	CF	IgM	Date	HI	CF
1. EK	13/7	19/7	<8	<4		2nd wk. Aug	128	8	+	1st wk. Sept	256	16
2. BM	1/8	8/8	16	<4		13/8	128	<4		25/8	128	<4
3. PR	28/7	7/8	<8	<4		20/8	128	<4				
4. MP	25/7	Last wk. July	<8	<4		4/8	32	<4		19/8	64	4
5. SA	5/8	15/8	16	<4		25/8	64	4		19/10	256	32
6. MA	20/8 (Died)	24/8	<8	<4		28/8	64	<4		4/9	1024	8
7. HC	23/8	26/8	<8	<4		31/8	64	<4		8/9	128	4
8. BJ	10/8	24/8	32	<4		31/8	128	<4		10/9	64	NSQ
9. CG	13/8	31/8	64	16	–	11/9	128	16		28/9	128	
10. McGT	24/8	1/9	32	<4		3/9	128	<4				
11. YE	14/8	18/8	<8	<4		2/9	32	<4		1/10	64	16
12. SJ	28/8	4/9	16	<4		18/9	64	<4		13/10	16	<4
13. CL	5/8 (Died)	12/8	64	<4	+	20/8	64	<4				
14. MD	27/8	1/9	<8	<4		6/9	32	<4		15/9	64	<4
15. KS	4/9	5/9	32	4		11/9	64	8				
16. KW	19/8	24/8	<8	<4		10/9	512	<4				
17. WC	17/8	21/8	8	<4	+					28/8	16	<4
18. WR	6/8	10/8	16	<4	+					16/9	64	16
19. BE	15/8	27/8	<8	<4						11/9	64	<4
20. VM	3/9	21/9	16	<4	+					2/11	128	<4
21. IP	27/8	31/8	<8	<4		14/9	16	<4	+	8/10	64	<4
22. FJ	3/9	8/9	<8	<4		10/9	16	<4		30/9	512	32
23. BP	1/10	9/10	16	<4	+					27/10	16	<4
24. McKD	14/9	13/10	16	<4	+					11/11	32	<4
25. GC	7/7	30/7	32	<4	+					14/9	32	<4

HI: Haemagglutination-inhibition test
CF: Complement-fixation test
NSQ: Not sufficient quantity

Epidemiological Study of Western Equine Encephalitis - Manitoba 1981

J.A. Eadie M.B.Ch.B., D.P.H.
B. Friesen M.D.

Preventive Medical Services
831 Portage Avenue
Winnipeg, Manitoba
R3G 0N6

Abstract

In 1981, the Province of Manitoba experienced an epidemic of Western Equine Encephalitis with 25 confirmed human cases. A study was carried out in order to get information about the clinical and epidemiological features of these infections in an attempt to identify causal factors. The study confirmed previous reports that farmers are at a high risk of being infected, their increased risk is likely due to a combination of personal and environmental factors. The results also suggest that exposure to infected mosquitoes is not the sole factor in determining whether or not illness occurs.

INTRODUCTION

Western Equine Encephalitis (WEE) is an arboviral infection, which occurs in man and horses, as a result of spill-over from the natural transmission cycle. Birds are the principal hosts and reservoir of WEE virus while mosquitoes, such as Culex tarsalis, are responsible for the transmission of the virus to non-infected birds, especially nestlings. If the environmental conditions are favorable to C. tarsalis and viruses are present in birds, then infections may occur in humans and horses, as "dead end" hosts, not a source of infection for others (McLintock 1975).

In Manitoba, WEE infection occurs sporadically, usually as isolated cases. Epidemics have occurred in 1941 (509 cases, 78 deaths), 1947 (81 cases), 1975 (14 cases) and 1977 (5 cases). In addition there have been equine epizootics in years when no human cases have occurred as for example in 1963 and 1966 with 173 and 51 horse cases of WEE respectively (Artsob and Spence 1977).

It was decided to carry out a case control study of the 1981 human cases of WEE in order to look at the various factors believed to be involved in the occurrence of infection. It was hoped that some association could be shown which would help in identifying causal factors and therefore allow primary and secondary prevention to take place. The personal characteristics of cases, such as clothing, repellant use, amount of time spent outdoors, occupation and the ecological surroundings such as presence of wild birds and location of nearest suitable mosquito breeding areas, were looked at.

A flaw in the selection of controls has prevented the analysis of the data collected by a case control method; instead, the cases will be described.

MATERIALS AND METHODS

Upon laboratory confirmation of a case of WEE (Sekla and Stackiw 1982), the attending physician was contacted and permission to approach the patient obtained. The case was then phoned and an interview arranged. It was requested that the spouse or another family member be present at the interview to validate the case's recall and provide additional information. Twenty-two of the 25 cases were interviewed including 2 who were interviewed by phone only, because of their distant residence. The 3 cases not interviewed were the 2 who had died and the infant; however, some information was obtained from their relatives. Additional information on the cases was obtained from the attending physicians, hospital records, and an ongoing study of central nervous system infections undertaken by the University of Manitoba.

Other family members, friends, neighbours and work associates were also interviewed after a case was seen. They were asked the same questions and bled to assess the presence or absence of specific WEE antibody.

RESULTS

Epidemic Curve

The Manitoba Arboviral Surveillance Committee (MASC) had recognized on July 23, the possibility of an epidemic of WEE. At this time the public was warned of the danger and advised to take personal precautions against mosquitoes. Human surveillance was intensified in order to identify any cases of WEE. The Provincial Epidemiologist notified physicians of the risk of a WEE epidemic through the Manitoba Medical Associations' newsletter.

The epidemic curve is shown (Fig. 1). The first case became ill on July 13, although he was not confirmed til August 13. He lived on a farm, 6 miles from a sentinel chicken flock and light trap which are located in a suburb of Winnipeg. The trap was the first one to show evidence of virus activity. The highest rate of onset of illness among humans took place during the week of August 17, to August 23. Five people became ill during this week. They had been infected sometime during the first half of August since the incubation period of WEE is 5 - 15 days (Beneson, 1980). Another 6 people became ill by September 6, when the epidemic curve ended. Two persons became ill after that time, one on September 14, and the other one on October 1. There was no obvious correlation between time of onset of illness and residence of the cases. The first 2 cases became ill within 2 days of each other, yet they lived 250 miles apart. The last person to become ill lived the furthest north of all 25 reported cases.

Residence

Five (20%) of the confirmed cases were from Winnipeg. Seven (28%) were from other cities, towns or villages, while the remaining 13 (52%) were from rural areas. The symptomatic case attack rate for Winnipeg was 0.91 cases per 100,000 residents. Residents of towns, villages and rural non-farm areas had attack rates of 2.4 per 100,000 persons. The rate for farm residents was the highest at 10.8 cases per 100,000 persons.

The geographical distribution of cases is shown (Fig. 2). There was no clustering of cases in any one area of the province. Cases occurred throughout Southern Manitoba.

Effect of Spraying

In 2 of the 5 cases from Winnipeg, the incubation period was prior to or during the aerial spraying; in the third cases, only 2 days of his incubation period occurred prior to spraying; the last 2 cases were infected 1 - 4 weeks after spraying and neither gave a history of exposure outside of Winnipeg during their incubation periods. It is not possible to compare the occurrence of cases before and after spraying elsewhere as the other areas had solitary cases only.

Age, Sex and Marital Status

The age and sex distribution of the cases is shown (Fig. 3). There were 21 males and 4 females. The median age for females was 71 years, while for males it was 40 years.

All the female cases were widows. They lived by themselves in their own homes. They looked after their lawns and gardens themselves, and as a result spent a large amount of time outdoors, especially in the mornings and in the evenings.

The age specific attack rate for cases is represented in Fig. 4. It shows a bi-modal distribution. The rates are high in the under 1 and again in the older age group. This interpretation must be qualified by the small number of cases in certain age ranges.

Occupation

The occupations of the cases are shown (Fig. 5). Farming was the most common occupation, with 10 full-time and 3 part-time farmers. As a result, farmers had the highest symptomatic case attack rate for WEE, i.e. 37 per 100,000. The median age of infection for farmers was 29 years old.

Outdoor Activities

The response of the cases to questions regarding whether the amount of physical activity or time spent outdoors had changed from the previous year showed that there had been little change. Fifteen (60%) had spent the same amount of time outdoors as the previous year, while 7 (28%) had spent increased time and 3 (12%) had spent decreased time outdoors.

Male cases had spent a greater amount of time outdoors than female cases. Males had a median exposure of 80 hours outdoors compared to a median of 45 hours for females. The greater exposure time for males is due to the long hours worked by farmers. If they are excluded, the median time for males falls to 45 hours, the same as females. The most frequent time period spent outdoors by cases was the evening. There was no correlation between the severity of illness and the amount of potential exposure. A history of unusual physically tiring exertion in the 24 hours before the onset of illness was given by 4 cases (16%); 17 cases (72%) felt their physical activity was not unusually different while no information was available on 4 cases.

Ecological Factors

The majority of cases had various sources of stagnant water that could serve as breeding grounds for C. tarsalis within 1 kilometer. Out of the 25 cases, 16 had ditches, 5 had rain barrels, 4 had old tires, 13 had sloughs, while 9 had a creek or river nearby.

Farm animals that could potentially attract mosquitoes or serve as amplifiers for WEE virus were also recorded. Nineteen cases had fowl, horses, cattle or pigs in the immediate vicinity of their residence. Of the remaining cases, 4 lived in cities, 1 lived in a town, but his dwelling was near a heavily treed park with a stagnant creek and 1 lived one block from a grain elevator and a slough-like lake; the latter 2 had large numbers of wild birds nearby. In assessing the amount of exposure to farm animals, it was noted that 8 had an association with 1 species of animal, 8 cases had an association with 2 species of animals, and 2 cases had an association with 3 species of animals.

Fifteen (60%) had been exposed to a species of birds - either chickens, ducks, turkeys or racing pigeons; 13 (52%) had been exposed to horses; 10 (40%) to cattle, but only 4 (16%) to pigs. Blood was obtained from 7 flocks of domestic fowl and 1 flock of racing pigeons. These birds were owned by either the patient or a nearby neighbour. Four of the flocks had antibodies to WEE by Hemagglutination - Inhibition (HI) testing. The number of birds with antibodies to WEE was 2 out of 25, 4 out of 25, 2 out of 3 and 2 out of 20.

Blackbirds and sparrows were the most common birds recalled by people as being present where they lived or worked. Sixty percent of the cases stated blackbirds had been present. Swallows were present in 44% and robins in 36% of the cases.

Family Members

Thirty-three family members were tested representing the household contacts of 16 of the cases. Of the other 9 cases, family members refused testing in one instance, in another the family members were not tested because of delay in the diagnosis, while the remaining 7 cases lived alone. Only 3 of these 33 contacts had antibodies to WEE by HI. All 3 had low titres of 1:8. One was a 45 year old native lady who had lived in the country all her life; the second was a 72 year old farmer who lived in the same rural area for 50 years; neither of these gave a history of recent or past infections that may have been WEE. The third individual was a 15 year old boy who helped his father, (a WEE case), on the farm; he had no history of illness. Both his older brother, who had also worked on the farm, and his mother had no antibodies.

Neighbours

Seventeen neighbours of 10 of the 25 cases were tested serologically. None had antibodies, even though 2 gave a history of illness clinically compatible with WEE, including 1 who was hospitalized with an aseptic meningitis confirmed by lumbar puncture. Viral cultures were not done on the spinal fluid. One week after the neighbour's illness, the WEE case was hospitalized with what was thought to be a recurrence of WEE infection. Aseptic meningitis was diagnosed following lumbar puncture and an Echo virus 6 was isolated from the CSF. It was not possible to test the neighbour's serum for antibodies to Echo virus 6.

Fellow Workers

Fifteen fellow workers of 12 of the cases had serum specimens tested for antibodies to WEE. The only one who had WEE antibodies was the hired man, who had worked for the farmer whose 15 year old son also had antibodies to WEE. The hired man had started work for the farmer after the farmer was ill. The hired man had previously worked nearby, training horses and dogs. Two of the horses he had been working with had become ill. The horses were lethargic and seemed to have stiff necks. They were examined by a veterinarian who felt their symptoms were related to being pulled behind a truck while training, rather than to a WEE infection; no blood was taken from the horses to test for antibodies to WEE. They had not been vaccinated.

Groups in WEE Endemic Areas

People from 3 different areas were tested serologically, in an attempt to identify additional cases of WEE. These areas were topographically favorable to mosquitoes and had shown evidence of WEE virus in the past. Two groups had no relation to WEE cases, while 1 group worked in a park where 1 of the cases lived.

The 2 groups unrelated to WEE cases consisted of people working in 2 marshland bird sanctuaries. These people were involved in various jobs such as guiding, research and maintenance. All were exposed, some more than others. At 1 marsh where a MASC trap had confirmed virus activity, only 1 out of the 15 people serologically tested had antibodies to WEE. This person had spent a great amount of time in various marshes in the preceeding 15 years as part of his employment as well as for recreation. His antibody titre remained static when a second serum specimen was tested.

In the second group, out of 25 persons tested only 3 had WEE antibodies and all 3 had been residents of the area for 15 years or more. Two of the 3 had low antibody titres of 1:8. The third had a titre of 1:64 which was found to be static when re-examined, 1 month later. None of the 3 had any symptom.

The third group consisted of 12 people who worked in a park near 1 of the cases. Three of the 12 had antibodies to WEE, but none had any symptoms. Two had low antibody titres of 1:8, while the third had an antibody titre of 1:32 by HI. They were middle-aged and all came from rural areas; 1 lived in a tent during the summer, another spent his evenings sitting outdoors and the third spent time fishing in a nearby marsh.

DISCUSSION

The symptomatic case attack rate on a province-wide basis was 2.5 per 100,000 persons, about half the rates reported for epidemics in Minnesota, North Dakota and California (Monath and Johnson 1980). Differences in the criteria used in the diagnosis of WEE and in the definition of the population at risk may account for some of the differences in the rates.

The greatest incidence in the 1981 epidemic occurred in the rural farm population, reflecting the fact that farmers were the most common occupation group to be infected. The increasing risk of infection with WEE as you progress from an urban area to a rural area has been previously reported (Hammon and Reeves, 1952). Past Manitoba epidemics have supported this.

The case attack rate of 10.9 per 100,000 persons living in rural farms in 1981 is much less than the rate of 79 per 100,000 reported in 1941 for Manitoba, excluding Winnipeg and its suburbs (Donovan and Bowman, 1942).

The rate for the 1941 epidemic was based solely on clinical diagnoses and as a result is likely inflated by cases of encephalitis due to other causes, however this itself does not explain the eight-fold difference between the rates in 1981 and 1941.

Another hypothesis is that in 1981, despite documented evidence of widespread WEE virus activity the risk of being infected was less than in other epidemics. This reduction in risk may be due to the environmental changes that have taken place since 1941, such as, for example, improvement in the quality of housing with better screening and with air conditioning in some homes. Nowadays, farm equipment is often enclosed with air conditioned cabs, which reduces the exposure of farmers to mosquitoes. Land drainage and the clearing of bush for agricultural purposes have also reduced mosquito breeding grounds and bird nesting areas. The switch from mixed farming to straight grain farming that has taken place since 1941 means there are fewer farms with animals attracting mosquitoes to the farmers.

Cases still occurred in farmers as not all farmers are able to afford the expensive enclosed equipment. The farmers infected in 1981 were young and just starting out. They owned older equipment with no cabs and they also tended to keep a few farm animals for additional income.

Also, all the farms visited had old tires, rain barrels, dugouts, sloughs, or other sources of stagnant water for C. tarsalis to breed in.

Local environmental factors are vital in the transmission of WEE. Equally vital in determining the risk of infection are some personal factors which may be illustrated as follows: 2 cases of WEE visited a farm which had a flock of 500 chickens kept outdoors in a pen; 10% of the flock had antibodies to WEE. One of the cases, who was a neighbour, had looked after the flock for a week, feeding them in the early morning and in the evening. The other case had visited 1 evening and spent less than 1 hour outside near the chickens. However, the hobby farmer, who owned the chickens, and his wife, had no antibodies to WEE even though they had a greater exposure than the 2 cases. Therefore, other personal factors must be involved such as individual attractiveness to mosquitoes. The style or colour of clothing, the use of perfume or mosquito repellants play an important role. The publicity campaign, warning people of the danger of WEE infection, focussed on these factors to reduce the risk of infection through personal protection. The apparent success of this primary prevention campaign may be seen in the fact that only 1 infant was infected in 1981. Parents appeared to be willing to take measures to protect their children from mosquitoes; however, they were reluctant to take the same measures for themselves.

The personal and environmental factors that have been mentioned already fail to adequately explain why among people with the identical exposures some develop infections and others do not. This point is illustrated as follows: 1 of the 1981 WEE cases was a market gardener. He and his wife worked together in their garden and had virtually the same exposure to mosquitoes in the time of peak C. tarsalis activity - the early morning and the late evening. There was no difference in the style of clothing or the use of repellant. However, the wife remarked later that mosquitoes have always bothered her less than her husband. They both became ill on the same day with fever, malaise and anorexia. The wife was ill for 2 days, then recovered, while her husband was hospitalized on the 3rd day with WEE encephalitis. The wife did not have antibodies to WEE by HI. or neutralization tests 1 month after her illnesses. This suggests that there may be a non-specific mechanism protecting some individuals. For example, guinea pigs that have been injected with mosquito salivary gland tissue and have developed antibodies require a higher infective dose of arbovirus than controls (Feinsod et al, 1975). This experiment suggests that some individuals may have built up a non-specific immunity response to mosquito borne disease, as a result of repeated exposure to mosquito bites.

The late occurrence of cases in 1981 is of concern to personnel involved in the surveillance and control of WEE infections. The location and timing of the last case suggest that either C. tarsalis remained active and seeking blood meals till late September or another species of mosquito such as Culiseta inornata was the vector. This means that certain aspects of the program to prevent human infections may need to be continued as long as there is evidence of mosquito activity. In certain years this may be late September.

The prolonged mosquito activity in 1981 may be the reason why cases still occurred in some areas after a single aerial adulticiding. For example, it was possible to document 2 cases occurring in Winnipeg, despite both aerial adulticiding and ground fogging. Aerial spraying does not confer absolute protection and is not an effective method of protecting the group at greatest risk, i.e. the farmers.

At this point in time, it is clear that there is no practical single method to prevent WEE infections. In the 1981 epidemic, prevention consisted of a combination of aerial spraying and personal protection. Aerial adulticiding and ground fogging are 2 examples of passive primary prevention. They require no effort by the individual. However, they do not confer absolute protection and are not feasible for the people at the greatest risk. Personal protection is active primary prevention. It can greatly reduce the risk of infection but it requires full perception of the danger of infection. Unfortunately, this is often difficult to achieve. Any preventive program using these methods should recognize these limitations.

Acknowledgements

We would like to recognize the guidance and suggestions that we received in this study. It is impossible to name all the people but we would like to especially recognize Dr. Sekla for her valuable help. We would also like to thank the attending physicians for their help. The patients must also be recognized for their willingness to participate in this study.

References Cited

Artsob, H., Spence, L. 1979. Arboviruses in Canada. In Hurstak, E. (Ed.) Arctic Tropical Arboviruses. Academic Press. New York, NY 39-65.

Beneson, A. 1980. Control of Communicable Diseases in Man. American Public Health Association. Washington, D.C. 413.

Donovan, C., Bowman, M. 1942. Epidemiology of Encephalitis - Western Equine Type Manitoba 1941. Can. Med. Assoc. J. 46:525-37. 1942.

Feinsod, F., Spielman, A., Waner, J. 1975. Neutralization of a Togavirus by Antivector Antisera. Ann NY Acad. Sci. 266:251-54.

Hammon, W., Reeves, W. 1962. Epidemiology of the Arthropod Borne Viral Encephalitides in Kern County, California 1943 - 1952. University of California Press. Berkley and Los Angeles, California p. 25.

Monath, T., Johnson, K. Diseases Transmitted Primarily by Arthropod Vectors. Last J. Ed. 1980. Maxcy Rosenau. Public Health and Preventive Medicine. 11th Edition. Appleton Century Crofts, New York, New York 332-334.

McLintock, J., 1976. The Arbovirus Problem in Canada. Can. J. Public Health (Suppl. 1). 67:8-11.

Sekla, L., Stackiw, W. 1982. Arbovirus Isolation and Serology in Manitoba. In Press.

FIGURE 1

WEEKLY EPIDEMIC CURVE
WESTERN ENCEPHALOMYELITIS
MANITOBA 1981

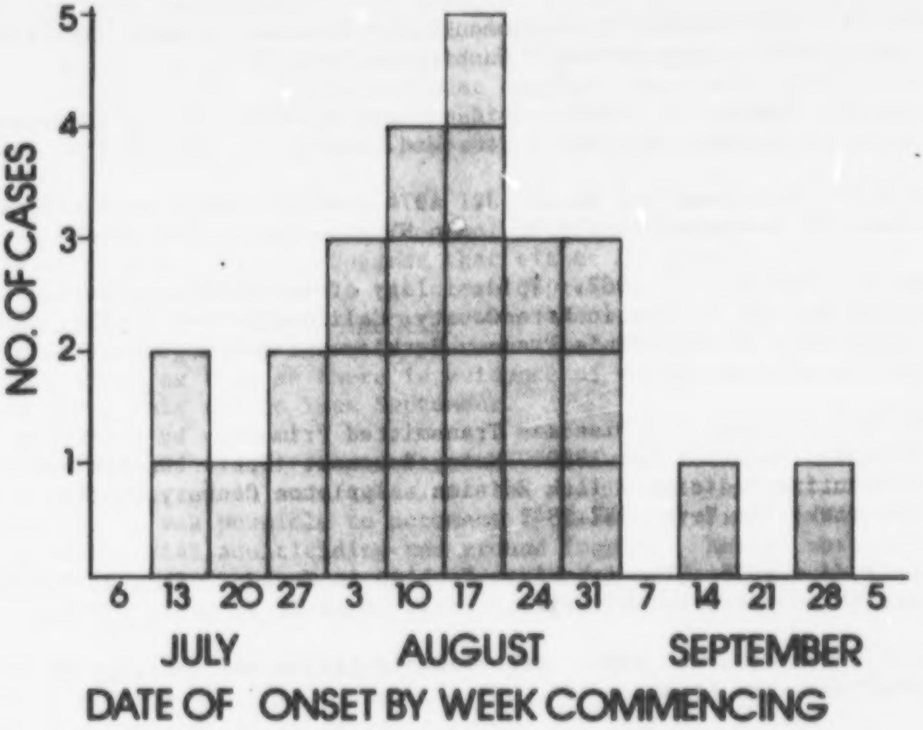


FIGURE 2

LOCATION OF CASES MANITOBA 1981

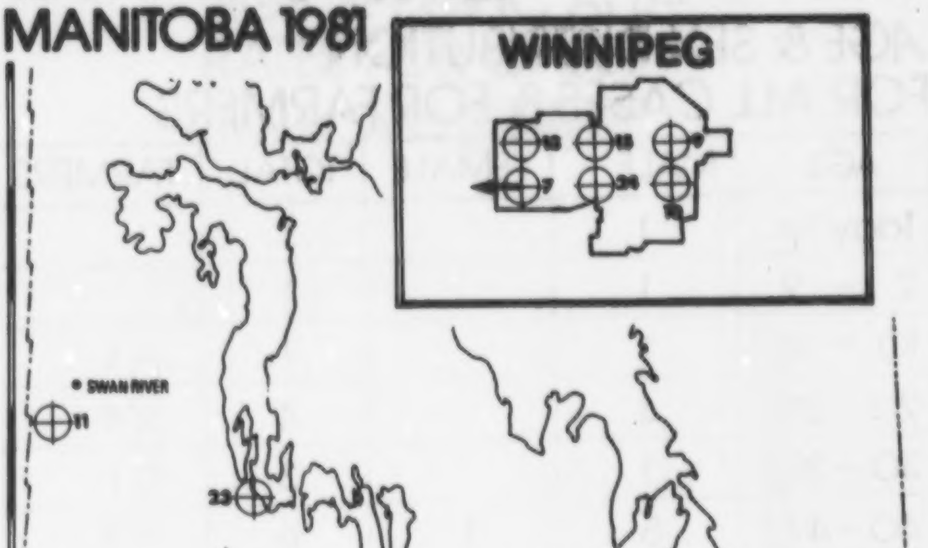

WINNIPEG
7
SWAN RIVER
11
23
12
DAUPHIN
LAKE WINNIPEG
LAKE MANITOBA
GIMLI
25
3
18
NEEPAWA
STONEWALL
SELKIRK
5
BEAUSEJOUR
PORTAGE LA PRAIRIE
VIRDEN
BRANDON
7
21
1
STEINBACH
14
6
MORDEN/WINKLER
22
100°

FIGURE 3

AGE & SEX DISTRIBUTION FOR ALL CASES & FOR FARMERS

AGE	MALE	FEMALE	TOTAL	FARMERS
1day-1yr.	1		1	
2 - 9	1		1	
10 - 19	1		1	1
20 - 29	4		4	4
30 - 39	3		3	1
40 - 49	5	1	6	2
50 - 59	4		4	2
60 - 69	O		O	
70 - 79	2	3	5	1
TOTAL	2	4	25	11

MALE	MEDIUM AGE	40yr.
FEMALE	MEDIUM AGE	71yr.

FIGURE 4

AGE SPECIFIC ATTACK RATES FOR CASES

MALES AND ■

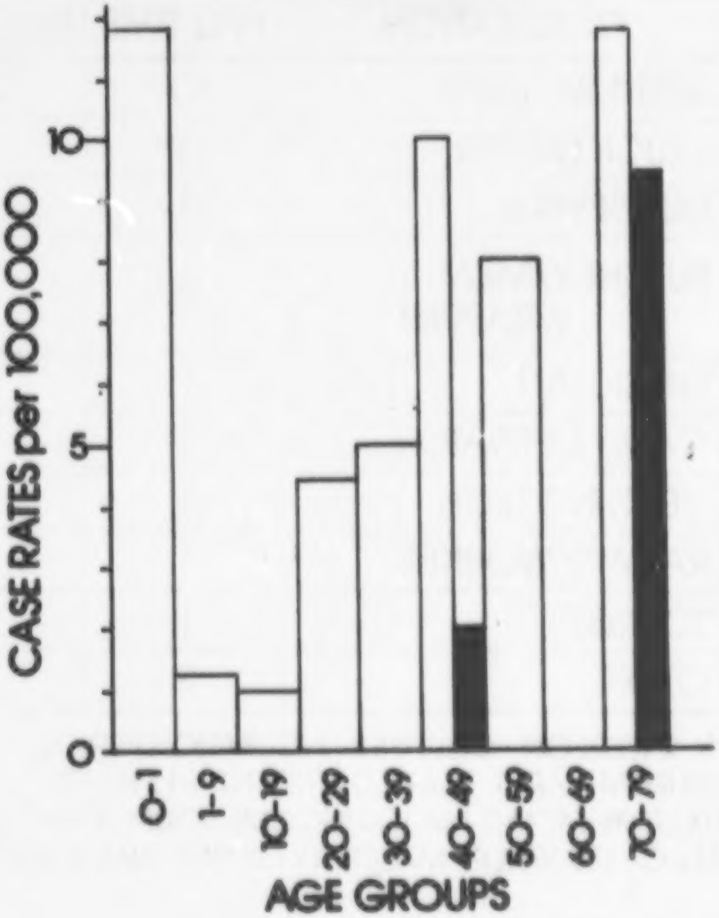


FIGURE 5

WESTERN ENCEPHALOMYELITIS MANITOBA 1981

OCCUPATION	FULL TIME	PART TIME
FARM WORKER	10	3
TRUCK DRIVER	3*	
HOUSEWIFE	3	
BUTCHER/MEAT WRAPPER	2	
PHYSICIAN	1	
CAMP OPERATOR	1	
VENDING TECH	1	
RAILWAY WORKER	1	
TOURIST	1	
OTHER	2	

* 2 WERE ON HOLIDAYS IN INCUBATION PERIOD
OTHER MEANS 1 8yr. OLD STUDENT + 1 INFANT
FULLTIME MEANS MAJOR INCOME SOURCE AND IN ALL CASES WHERE MAJORITY OF TIME WAS SPENT.

CHAPTER VII:

Vector Control

Insecticides Registered for Biting Fly Control in Canada

J.E. Hollebone, M.S., Ph.D., D.I.C.,
Pesticides Division, Agriculture Canada, Ottawa, Ontario
K1A 0C6

Abstract

Pesticides registered for use in biting fly control programs in Canada are discussed in terms of rate, application techniques, environmental persistance and toxicity. The paper is intended as an aid for the non-entomologist charged with choosing appropriate mosquito control products.

Biting fly control products available for use in Canada undergo a pre-registration review to determine that the product will be effective against the pest and safe under the conditions of use to user, bystander and environment. This involves an examination of scientific field studies conducted at several locations across Canada, and agreement from federal consultants in Health and Welfare, Environment Canada and Fisheries and Oceans that the product meets safety criteria.

Under the Pest Control Products Act, product labels spell out the conditions for use and disposal, environmental limitations, and first aid and safety precautions.

Two broad categories of products are registered for outdoor mosquito control in Canada: i.e., larvicides which are confined to growing populations in aquatic breeding sites, and adulticides used in the terrestrial adult habitat.

Larvicides

The larval and pupal stages of all mosquitoes develop in water. Insecticides used at this stage in development can be very effective in breaking the life cycle and in reducing future adult populations. Approved larvicides should be used in conjunction with physical controls or when permanent control measures (such as drainage, filling of low areas or diking) are not feasible.

In Manitoba, larvicides can be used effectively in the snowmelt ponds which are home to spring-breeding Aedes spp. and in midsummer aquatic sites which harbour the vector of Western Equine Encephalitis (WEE), Culex tarsalis, summer Aedes spp. and other mosquitoes.

The of application is critical in a management program for greatest effectiveness. Larvicides should be applied when the majority of "wrigglers" are in the 2nd-3rd larval instar. If applied after pupae are present, many products will not be effective. Larvicide treatments may need to be repeated for successive generations of mosquitoes. The timing of such treatments must be determined by monitoring the breeding area for developing populations. Larvicides should not be applied when larvae are not present.

There are several criteria to be considered in choosing a larvicide. Among these are formulation type, cost, mode of action, environmental effects, residual activity, environmental persistence, temperature dependency of active ingredient, safety to the user and ease of handling. Although some of these are self explanatory, others will be discussed briefly in the following text.

Larvicides are formulated as granules, emulsifiable concentrates, wettable powders, and ready-to-use briquets. Granular formulations have some advantages over liquid formulations. Many mosquito breeding sites are surrounded by vegetation of varying thickness - which may affect ease of application.

Liquid formulations generally work well in areas of low vegetative cover; granules more readily penetrate thick vegetation, tend to drift less than liquids and are more effective in areas with some vegetative cover. On the other hand, liquid formulations are often less expensive, and the user usually also must weigh economic considerations. The ready-to-use briquet has proven to be convenient for small breeding sites such as ditches or catch basins.

Slow release formulations are now available for a number of products, and are both acceptable environmentally and economically because of the increased interval possible between treatments.

The active ingredients registered for larval mosquito control (Table 1, 2), along with their relative toxicities, formulation, application types and rates of application are given below.

The older larvicides such as mineral oil, malathion and methoxychlor depend upon relatively high rates of application to effect control. For example, the recommended 22-55 L/ha required for effective control with mineral oil to the very low rates (15-55 mL or g/ha) for the organophosphates temephos or chlorpyrifos. (Table 2). The trend has been in recent years towards the development of products with effective but lower rates of application.

The mode of action of the various larvicides differs. Mineral oil formulations have the advantage of being ovicidal and pupicidal as well as larvicidal. The mode of toxic action is thought to be a combination of suffocation and poisoning from the volatile components of the oils. However, oils may be ineffective in areas of dense vegetation if larvae should find a breathing area in the film break around aquatic plants. The organophosphates, temephos and chlorpyrifos, are among the most frequently used larvicides but are not particularly effective against pupae or as ovicides. Temephos tends to be used for situations requiring a single application, due to its short residual activity, chlorpyrifos in situations requiring residual control.

The newest larvicides are the insect growth regulators such as methoprene (Altosid) and diflubinzuron (Dimilin), and the newly registered biological agent, Bacillus thuringiensis ser H 14. These are narrow spectrum biocides, specific to relatively few other organisms besides the target organism. Methoprene is a synthetic insect juvenile hormone, which delays maturation by deforming the pupa and preventing viable adults from emerging. Diflubenzuron blocks a final step in chitin synthesis, prevents cuticle hardening, and causes death by disrupted water balance. The bacterium Bt H14 contains a crystal endotoxin which when ingested by a feeding larva is activated to toxins which rupture the mosquito gut, causing rapid disruption of the haemocoel, paralysis and death. Dimilin will affect any arthropod, as all have chitinous exoskeletons; methoprene is toxic to Daphnia, Bt H14 has shown selective toxicity only to nematocerous flies closely related to mosquitoes. None of the three has shown any effects on other aquatic life such as tadpoles, fish, birds or mammals. Bt H14 has not yet been used in Canada, but gives the promise of good control coupled with minimal environmental effect.

Residual activity is important in choosing a larvicide. The low residual activity of malathion, mineral oil, temephos, chlorpyrifos and methoxychlor is 1-3, 4-5, 2-4, 10-14 and 10-14, day 5, respectively. Temephos, therefore, would be effective for single applications, such as for temporary ponds containing spring *Aedes* spp. In summer ponds where generations follow in rapid succession, a residual insecticide which would reduce the necessity of frequent repeat applications, such as chlorpryrifos, would be the larvicide of choice. The growth regulators tend to be non-persistent: i.e. methoprene, in hot sunny weather has a field life of 2-3 days, but in cooler periods, it has a field life of over a week. The development of a slow release briquet, however, allows methoprene to be used effectively in small confined areas such as catch basins or transformer vaults for up to 30 days. *B.t.* H14 has an effective life of 24-48 hours. Its fate in the environment after this time is not satisfactorily understood. There are some indications it may hydrolize in water, others that it settles out onto bottom sediments.

Temperature dependency is another factor which has an important bearing on efficacy. Malathion does not appear to perform well in cool temperatures. Recently Helson and Surgeoner (1982) have shown that the efficacy of temephos is also temperature dependent. Given that all larvicides registered are effective in warmer temperatures (i.e. greater than 15°C) residual activity, environmental and manpower considerations become determining factors in choosing the appropriate larvicide for use. At cooler temperatures, efficacy-temperature relationships become important.

Finally in the day of public concern over the environment, the environmental effects of a larvicide may affect final choice. Methoxychlor and chlorpyrifos are toxic to fish at labelled rates. These, therefore should not be used in fish productive waters. Chlorpyrifos and dimilin are toxic to crustaceans. This may not be important in treatment of temporary pools, but care should be taken to avoid contamination of running water. At levels just above registered rates, fenthion can be toxic to birds. The insect growth regulators, have shown very low environmental impact to non targets are most environmentally acceptable. (Table 3).

Adulticides

Adulticiding becomes necessary when larviciding is not practical, (e.g., after adverse weather conditions which may make access to breeding sites impossible, or manpower limitations of covering the treatment area). Because adults often migrate into control areas from untreated locations, adulticiding can only be as effective as the size of the area treated, weather conditions and the type of chemical and equipment used.

Most adulticide programs in Canada use a combination of residual sprays, thermal foggers and cold Ultra Low Volume (ULV) application of chemical. Thermal foggers are being gradually phased out as equipment wears out. Thermal foggers, which are available as both portable and truck mounted units, heat a mixture of insecticide and diesel oil diluent and dispense it by a powerful air blast system as a dense drifting fog. Most fogs do not persist signficantly on substrates as residual spray droplets.

The expense and time involved in carrying and handling the diesel oil carrier are major disadvantages of thermal foggers, which have largely been overcome in the ULV cold foggers. Product is used undiluted and unheated in these machines, either in a portable or truck-mounted unit. Precise regulation of droplet size to 5-50u Mass median diameter (MMD) and a metered flow rate calibrated against vehicle speed allows an inconspicuous cloud of minute droplets to be emitted in a very low volume (15-45 mL) per hectare. Five insecticides have been registered for ULV application by ground equipment: i.e., propoxur (Baygon), chlorpyrifos (Dursban), malathion (Cythion), pyrethrin and resmethrin. The same five are also registered for thermal fog use as well as dichlorvos (Vapona), fenthion (Baytex), and methoxychlor. (Table 4).

Most of the products registered for ground sprays are also available for aerial use with conventional fixed wing aircraft or helicopters: i.e., propoxur, chlorpyrifos, fenthion, malathion, naled and methoxychlor. For ULV aerial application, two choices are available: malathion or propoxur. (Table 5).

The choice of an adulticide depends upon similar criteria to those used for choosing a larvicide. It must be effective at the time of optimum application (early morning or late evening), have some residual activity but not be persistant in the environment, and must minimize undesirable environmental effects. For these reasons malathion, propoxur, chlorpyrifos and methoxychlor are the most frequently chosen adulticides. Propoxur combines good efficacy with 5-7 days residual activity, Dursban 14 days, malathion 3, methoxychlor 7-10 days activity. Propoxur is effective in cool temperatures, whereas malathion works best above 15°C. Neither is as toxic to fish as Dursban or methoxychlor, and both have been widely used across Canada as adulticides. All have some toxicity to bees, and spraying programs should try to minimize bee exposure. Resmethrin is the newest adulticide registered. It combines high toxicity to mosquitoes with low residual action. It is extremely toxic to fish. This, plus its presently high cost, may affect its widespread use in Canada.

Finally, other products are available, although not registered specifically for biting fly control programs. Several pesticides are registered for fly control on livestock (or around barns and farm buildings): ronnel, dichlorvos, malathion and various pyrethrin mixtures.

In many locations, especially remote rural areas, there are no practical means of controlling mosquitoes. In such locations, any of the registered personal repellents will provide protecion to humans if used correctly. Diethyltoluamide is one of the best repellents. For those who have sensitive skins or skin allergies, a repellent jacket which can be worn over the outer clothing is available.

In conclusion, there are many products available for chemical and now, biological, control of mosquitoes in biting fly programs. The actual choice of which to use must be made within the framework of an understanding of the life cycles of the pest insect, the time and parmeters of application, the weather pattern, the equipment available, and the nature and action of the candidate products for chemical control.

References Cited

Helson, B.V. and Surgeoner, G.A., 1982. Unpublished manuscript. Effect of temperature and stage of development on susceptibility of Spring Aedes spp. larvae to Temephos.

TABLE I

Relative toxicities of technical active ingredients used in mosquito control products.

Active Ingredient	Acute Oral LD_{50}, rat (mg/kg)	Acute dermal LD_{50}, (mg/kg)
Bacillus thuringiensis ser H 14	2,670 **	rat 6,280**
Chlorpyrifos	97 - 276	rabbit 2000
Dichlorvos	56 - 80	rat 107
Diflubenzuron	4,640**	rabbit 2000**
Fenthion	250 - 300	rat 330
Malathion	1,375	rabbit 4100
Methoprene	34,600**	rabbit 3000**
Methoxychlor	6,000	not available
Mineral Oil	10,000**	rabbit 3160**
Naled	430	rabbit 1100
Propoxur	95 - 104	rat 1000
Pyrethrin	1,500**	rat 1800
Resmethrin	4,230**	4640**
Temephos	8,600	rat 4000**

* Values from Agriculture Canada Files and Pesticide Dictionary Section D, 1979, Farm Chemicals, Meister Pub. Co., Ohio 44094

** Highest rate tested

TABLE II

LARVICIDES REGISTERED FOR MOSQUITO CONTROL IN CANADA

ACTIVE INGREDIENT	FORMULATION TYPE	RATE OF APPLICATION ai/ha	TYPE OF APPLICATION	COMMENTS
Mineral Oil (Flit MLO)	EC	22-55 L	Ground or Aerial	Rate depends upon vegetation, surface conditions.
Malathion (Cythion)	EC, SN	550 mL in oil or water	Ground	Oil base may cause slight phytotoxicity.
	SN	325-500 mL in 250-175 mL oil	ULV Aerial	Apply per label instructions
Methoxychlor (Methoxol)	EC, SN	200-275 mL in oil or water		Do not apply to water containing valuable fish.
Fenthion (Baytex)	EC, GR	55-110 g	Ground or Aerial	Use higher rate if pools are >30 cm deep.
	WP	55 g		May be toxic to birds at high rate. If treated pasture, do not feed to animals for 3 days.
Temephos (Abate)	GR, EC	55-100 g^1	Ground, Aerial	1. Use for water of low organic content.
		100-225 g^2		2. For water of high organic content.
		550 g^3		3. For heavily polluted water.
Chlorpyrifos (Dursban)	EC	15-55 mL in oil	Ground, Aerial	1. Use low rate with low vegetative cover or organic content.
	GR	27.5-55 g	Ground, Aerial	2. Use high rate for high vegetative cover or organic content. 3. High rate toxic to fish and crustacea. 4. 2 week treatment interval.
Pyrethrin and technical Sulfoxide	SN	PYR 0.335 + SFO 1.778	Ground	
Methoprene (Altosid)	SU	29-36 mL in water	Ground or Aerial	
	TA	32 mg/m^2 1	Ground	1. Apply every 30 days.
Diflubenzuron (Dimilin)	GR,WP	27.5-45 g in water	Ground or Aerial	1. Do not use fish or shellfish for food. 2. Do not use on treated crops for food.
Bacillus thuringiensis var. H 14	WP	0.28-1.1	Ground	1. Do not use in potable water

TABLE III

ENVIRONMENTAL EFFECTS OF SOME BITING FLY CONTROL CHEMICALS

ACTIVE	ENVIRONMENTAL TOXICITY TO LC$_{50}$ ppm at 48 hr. Invertebrates	Fish	LD$_{50}$ Birds	RESIDUAL ACTIVITY	PERSISTENCE
Bt H14	toxic to simuliids dixids chironomids ceratopogonids but not to other aquatic invertebrates	none reported to bluegill rainbow trout 30 day exposure	none reported mallards	24-48 hr.	48 hrs.
Chlorpyrifos	.0010 stone flies	highly toxic .053 rainbow trout 0.020 shiner 0.03 green sunfish	mod.-toxic 70-80 mallard 80 Can. geese 25-50 lesser sandhill crane	10-14 days	80% disappears in 5-15 days, 90% cleared by 30-35 days, 90-99% lost from plants in 10 days LC$_{50}$ = 80 days sand 16 days muck soils
Dichlorvos	.001 Gammarus 0.023 stoneflies	0.7 bluegill	7.8 mallard	3-4 days	subject to rapid hydrolysis persists in one study for 67 days @ 20°C
Diflubenzuron	0.7 ppb to shrimp	135 (96hr) Lepomis 140 rainbow trout	5000 mg/kg mallards	24 hrs.	in soil 3-7 days; in water degrades within 24 hrs. to 4-chlorophenyl urea
Fenthion	.003-.004 Daphnia 0.15 amphipods 0.07 stoneflies	0.80 brown trout 1.3-1.8 various fish	5-9 mallard 12.0 Can. geese	4 days	degrades 90% in 2 weeks in river water, pH 7.3 100% in 4 weeks

TABLE III (cont'd)

ENVIRONMENTAL EFFECTS OF SOME BITING FLY CONTROL CHEMICALS

ACTIVE	ENVIRONMENTAL TOXICITY TO LC_{50} ppm at 48 hr. Invertebrates	Fish	LD_{50} Birds	RESIDUAL ACTIVITY	PERSISTENCE
Malathion	.003 stoneflies .0010 waterfleas .004 amphipods	0.02-.3 trout, bass 6.59 carp 10.7 goldfish	1485 mallard	1-3 days	instant hydrolysis in alkali completely degraded in river water in 4 weeks Lt_{50} 1 week to 4 weeks
Methoprene	toxic to Daphnia but not to other invertebrates	no effect on fish, amphibians	2000 mallards	up to 25 days slow release briquet N.D. after 6 days 10% SR.	rapidly photo-degraded 100% in 7 days to 50 minor photolysis products, halflife in soil 10 - 14 days
Methoxychlor	.0047 Gammarus	highly toxic .0072 rainbow trout -.2 bass	2000 mallards	7-14 days	rate is directly related to content of organic matter less than 50% detected after 300 days Lt_{50} - 21 -28 days
Mineral Oil	at 22-35 L/ha no kills to simids, shrimp, tadpoles, frogs, but some to dytiscids hydrophilids, notonectids	at 96 hr. 3000 ppm salmon threshold tox.= 1000 pm or 4000 L/ha	not toxic at use levels LC_{50} 10,000 ppm ducklings	4-5 days	4-5 days, chemical and biological degradation
Propoxur	.005 amphipods 0.11 stoneflies	0.025 minnow	40-60 sandhill crane 6 Can. goose 119 mallard 3.6 house finch	4-7 days	photo-degrades on plants Lt_{50} 0.5-5 days in soil Lt_{50} 9 - 25 days in river water 50% hydrolysis in 1 week, N.D. at 8 weeks
Temephos	0.0020 shrimp .96 amphipods	25 Lepomis 200 bluegills 200 bass	79.4 mallard 42 blackbird 270 partridge	3-4 days	decreases to less than 1 ppb in water by 14 days

TABLE IV

ADULTICIDES REGISTERED FOR MOSQUITO CONTROL IN CANADA

GROUND APPLICATION

ACTIVE INGREDIENT	FORMULATION TYPE	RATE OF APPLICATION ai/ha	TYPE OF APPLICATION	COMMENTS
Propoxur (Baygon)	SN	17.5-27.5 ml	Space Spray Thermal Fogger Cold Fog ULV aerosol generator	1. May be diluted with oil. 2. Toxic to bees.
Chlorpyrifos	SN, EC	27.5-55 mL 27.5 in 2 L oil 5.5-11 mL	Hand, power sprayer Mister Thermal Fogger ULV cold Fogger	Do not apply to water. Follow label instructions.
Dichlorvos (Vapona, DDVP)	SN	50-150 mL in oil	Space Spray, Foggers	
Fenthion (Baytex)	SN	55-110 mL in oil	Thermal fogger	
Malathion (Cythion)	EC EC, SN	550 mL in oil or water 98 in oil or water	Space Spray Fogger	
Methoxychlor	EC EC	200-275 mL 35-70 mL	Space Spray Thermal fogger	
Naled (Dibrom)	SN EC	175 mL in oil 110-275 mL in water	Thermal fogger Mist blower	May spot painted surfaces.
Pyrethrin mixtures	SN	As per label	ULV foggers thermal foggers backpack sprayers	
Resmethrin	SN	7.5 g in 400 mL ai	ULV backpacks ULV foggers	
Mixtures Fenthion + Lethane Malathion + Lethane Methoxychlor + Lethane	SN	As per labels	Thermal fogger Mist sprayers	

TABLE V

ADULTICIDES REGISTERED FOR MOSQUITO CONTROL IN CANADA

AERIAL APPLICATION

CONVENTIONAL AERIAL APPLICATION	FORMULATION TYPE	RATE OF APPLICATION ai/ha	COMMENTS
Propozur (Baygon)	SN	32.5-125 mL in oil	Use higher rate for dense vegetation. Toxic to bees.
Chlorpyrifos (Dursban)	EC	27.5-55 mL in oil	At high rate fish and crustacea may be killed.
Fenthion (Baytex)	EC, SN	55-110 mL in oil	Toxic to birds at high rate. Do not graze or cut for feed for 3 days post application.
Fenthion and Lethane	SN	FET 55-110 mL +LER 325-650 mL in oil	
Malathion (Cythion)	EC	550 mL in oil or water	Toxic to bees and fish.
Methoxychlor	EC	55-270 mL in oil or water	Apply with care near fish ponds.
Naled (Dibrom)	EC, SN	55-270 mL in oil or water	Toxic to bees, fish, crustaceans.
ULV APPLICATION			
Propozur (Baygon)	SN	32.5-125 mL	Toxic to bees.
Malathion (Cythion)	SN	32.5-600 mL	Toxic to fish and bees.

Alternatives to Chemical Control of Mosquitoes in Manitoba

M. M. Chance, M.Sc., Ph.D.

Canada Biting Fly Centre, University of Manitoba, Winnipeg, R3T 2N2

Abstract

Alternatives to chemical methods for the control of mosquitoes include source reduction, biological control, and host protection. At existing levels of development, these strategies can provide at best only partial control of mosquitoes in Manitoba. In general, their use is compatible with chemical methods and their integration into all control programs is recommended.

Mosquitoes have 4 stages in their life cycle: i.e., egg, larva, pupa, and adult. Female mosquitoes lay their eggs on moist soil, vegetation or the water surface of their breeding sites. Both larva and pupa are aquatic. Larvae of most species feed by filtering small organic particles. Pupae do not feed. Adult females of biting species need to obtain blood in order for them to complete development of their eggs. When not actively seeking a blood meal, mating, or dispersing, adults rest among vegetation or other cool, moist locations.

Mosquitoes breed in all types of standing water, including the following: i.e., permanent and temporary ponds; sloughs; irrigation ditches; impoundments; reservoirs; waste water and sewage ponds; snow-melt and floodwater pools; and natural and artificial containers such as tree holes, rain barrels, cemetery urns, bird baths, and discarded tires. In Manitoba, the most severe nuisance species are the snow-melt and floodwater temporary pool-breeding *Aedes*. *Culex tarsalis* Coquillet, the known vector of WEE, breeds in a variety of sites including permanent and temporary pools, and artificial containers. Adult females of this species overwinter hibernating in culverts, animal burrows, rock piles and other sheltered areas (Shemanchuk 1965). Most other Manitoba species overwinter in the egg stage.

Programs to control mosquitoes are usually directed against larvae. Larviciding programs eliminate mosquitoes before they bite. In addition, the larval stage is most vulnerable to attack because larvae are localized in their breeding sites.

Chemical control is the most effective approach today for controlling mosquitoes, both in terms of eliminating mosquitoes and cost effectiveness. However, chemical methods require repeated applications and do not provide permanent control. In addition, the potential environmental hazards associated with the application of insecticides and the possible development of resistance among target species necessitates development of alternative control strategies[1].

Three approaches offer alternatives to chemical control: i.e., Source reduction is the elimination or manipulation of breeding sites, rendering them unsuitable for mosquito survival. It is also called physical, mechanical, or cultural control, or environmental manipulation. Biological control is the use of organisms (i.e., predators, parasites, or the pest species itself) to reduce numbers of the pest organism. Host protection is the prevention of mosquito bites by the provision of chemical or physical barriers. Unlike the aim of other control strategies, the aim of host protection is not the reduction of pest abundance.

SOURCE REDUCTION

The source reduction of mosquitoes is most successful when directed against larvae. Breeding sites can be destroyed by improving soil permeability, drainage, ditching, diking, or by land-filling or leveling. Removal of emergent and marginal vegetation and banking will destroy shallow productive breeding areas in permanent sites (Mulhern 1975). Destruction of adult resting sites is possible by removal of vegetation but is successful only at a very localized level.

The environmental impact of source reduction of natural breeding sites (e.g., a waterfowl marsh) is drastic and may exceed that of chemical control. However, the source reduction of man-made breeding sites in urban and agricultural areas, and along highways and railways is environmentally acceptable both with respect to the manipulation of the environment and to the reduction of the use of chemicals.

Because of the extent and nature of breeding sites in Manitoba, source reduction is not practical on a large-scale basis and offers only a reduction of numbers of sites requiring control. It is therefore only a partial alternative to chemical control. Although it is expensive to implement and to maintain, it does provide long term solutions.

BIOLOGICAL CONTROL

The appeal of biological control is the potential for permanent, selective mosquito control, that is, maintenance of pest species below tolerance thresholds by self-sustaining and environmentally harmless means. It offers a "natural" means of controlling mosquitoes, with an inferred and sometimes undeserved attribute of involving less human disruption of the environment.

Biological control may be achieved by one or more of the following approaches; i.e., (i) the conservation or enhancement of natural populations of predators or parasites, (ii) the inoculation of endemic or exotic predators or parasites, (iii) the mass release of predators or parasites into habitats in which they do not normally occur or occur only at low levels, or by (iv) the use of the pest itself (e.g., genetic manipulation).

---

[1] Resistance has not occurred in Manitoba so far.

Mosquitoes form a normal part of the diet of a variety of predators. Several species of larvivorous fish have been used for mosquito control. The mosquito fish, *Gambusia affinis* (Baird and Girard), has been employed extensively in warm climates (Laird 1981, McHugh *et al.* 1968) and strains of this fish have been developed which can survive in northern U.S. (Gall *et al.* 1980, Mulhern 1975). An introduction into hot spring pools in Banff, Alberta, survived over 50 years (Mail 1954). Yet field releases of *Gambusia affinis* have sometimes failed (e.g., where vegetation provides shelter to mosquito larvae). Also, this fish may, itself, become a pest. Its environmental impact can be as severe as that of chemical control and its introduction into permanent and semi-permanent waters has been discoraged (Ebeling 1978, Mulla *et al.* 1979). However, it can be useful in artificial habitats (e.g., dugouts and ornamental pools).

Other larvivorous fish have been used successfully, e.g., the guppy, *Poecilia reticulata* Peters; the three-spined stickleback, *Gasterosteus aculeatus* L.; and the fathead minnow, *Pimephales promela* Rafinesque (Ebeling 1978, Laird 1981). The stickleback has been investigated for mosquito control in British Columbia (Regan and Rosberg 1980) and the fathead minnow in Manitoba (Ellis 1980a). Although a hardy and locally occurring species capable of withstanding exposure to pesticides, the fathead minnow apparently cannot survive winter freezing or shallow warm waters in the spring (Ellis 1980b).

The potential for fish as control agents in Manitoba is limited to use only in some permanent breeding sites. Fish are unsuitable for controlling spring mosquitoes breeding in temporary pools. Stocking of shallow, permanent breeding sites would require costly indoor maintenance over the winter. In sites deep enough to permit survival under the ice, the fish may starve when prey species are scarce or feed on beneficial insects.

Insectivorous birds are often considered as control agents of adult mosquitoes. There have been many attempts to increase local populations of purple martins and other swallows. However, these birds, as well as insectivorous bats, feed on a variety of insects including some mosquito predators (Kale 1968). Mosquitoes are not a significant part of swallows' diets and there is no evidence that these birds significantly reduce mosquito populations (Hocking 1972, Kale 1968). Bats are generally too scarce to reduce mosquito populations in Manitoba.

A variety of predatory insects, hydra, and planaria have been studied for mosquito control (Bay 1972, 1974; Laird 1981). The use of insectan predators for mosquito control is not yet feasible. They do not occur naturally in sufficiently high numbers to reduce pest populations significantly. Mass releases are not yet possible because there are no adequate rearing techniques. In addition, because they grow more slowly than mosquitoes, predators tend to be unsuitable as control agents of larvae in temporary pools.

Hydra may be appropriate predators for temporary pool mosquitoes in Manitoba. Hydra are efficient predators of mosquitoes, they can survive harsh environments, and they can be mass-produced. In California Hydra have been used successfully against *Aedes* species and *C. tarsalis* (Cress 1980). Releases of planaria in Ontario reduced populations of mosquito larvae in catch basins by an overall 70% (George 1978) and simulated field releases in California reduced populations by 90% (Cress 1980). Planaria are probably effective only in small, contained sites where prey are confined.

Several aquatic plants have larvicidal properties (Angerilli 1980, Ebeling 1978). However, practical results of their use in mosquito control have not been demonstrated.

Parasites offer greater success than do predators as biological control agents. Recently a bacterium (*Bacillus thuringiensis* H-14 (var. *israelensis* de Barjac), "*Bti*") became the first biological agent for biting flies to be commercialized. *Bt.*H-14 is effective, selective, biodegradable, and easily mass-produced and handled (Garcia *et al.* 1980; Laird 1981, 1982). It is effective over a wide range of temperatures including cooler temperatures encountered in prairie spring breeding sites. Its environmental impact is minimal when compared to that of chemical larvicides. Its use should be encouraged. However, its short residual life and lower efficacy in highly organic waters (and resultant greater cost) may limit its use in Manitoba.

Bacillus sphaericus Neide is another bacterium that rates serious consideration as a mosquito control agent. *B. sphaericus* provides acceptable control under experimental conditions (Laird 1981) but is less effective than *Bt.*H-14 (Wraight *et al.* 1982).

Among the fungi, species of *Coelomomyces* and *Lagenidium* are most likely candidates for use as control agents (Chapman 1974a, Roberts 1974). *Coelomomyces* species are widespread and relatively specific. Some species can be mass-produced and provide high rates of infection in natural populations (Frederici *et al.* 1980). In field tests in California, inoculations of *Lagenidium giganteum* Couch eliminated *C. tarsalis* populations in 5 days and survived in same locality for at least 4 years (Laird 1981). Fungi, isolated from mosquitoes in the Canadian prairies, include species of *Coelomomyces* (Laird 1982, Shemanchuk 1977, Taylor *et al.* 1980). The potential for fungi that are parasitic in prairie mosquitoes is being actively studied in Manitoba and Alberta.

Nematode worms produce very high levels of infection in mosquitoes

(Chapman 1974b, Trpis *et al.* 1968). Two species, *Romanomermis nielseni* (Tsai and Grundmann) and *Romanomermis culicivorax* Ross and Smith, can be cultured, the latter in large numbers (Chapman 1974a; Laird 1981, 1982). *R. culicivorax* has little effect on non-target species (Mulla *et al.* 1979) and is close to becoming commercially produced (Laird 1982). Field releases of *R. culicivorax*, a warm-water nematode, in Manitoba for control of temporary pool mosquitoes demonstrated the difficulties in using these nematodes under prairie springtime temperatures (Galloway 1975).

Viruses that are pathogenic to mosquitoes are also recognized as potential control agents (Chapman 1974a, Frederici 1974) but, to date, very little research has been directed towards functional control programs. Techniques for culturing viruses in adequate amounts and protocols necessary to assess the safety and environmental acceptability of viruses are not yet available. Similarly, protozoan parasites of mosquitoes have been investigated (Chapman 1974a, b) but the lack of information on their biology and transmissions and on culturing techniques make it impossible to assess their potential for mosquito control.

Genetic control strategies offer potentially effective and safe means of mosquito abatement (Rai 1972, Asman *et al.* 1980). Although some techniques have been developed for genetic manipulation in laboratory-reared strains, none have been developed to the stage where they can be implemented successfully.

CONCLUSIONS

Biological control is not a panacea. Technological development is still experimental rather than operational. The major problems include shortcomings in mass-rearing techniques, standardization of efficacies and monitoring procedures. Biological agents have the potential to reduce populations to below nuisance levels but they will not break the transmission cycles of diseases. They rarely eradicate pest populations. They do not offer an alternative to chemicals for rapid, emergency control. They are suitable for sustained programs and they tend to be environmentally more satisfactory.

The implementation of either biological control or source reduction programs requires thorough investigation. Each control problem needs a 'custom-made' rather than an 'off the rack' approach. Both strategies provide approaches complementary to chemicals and should be included in control programs wherever possible. The immediate gain to be made from these alternatives is a reduction in the amount of, and sole reliance on, chemicals used for control programs.

When considered as an alternative to chemical methods, biological control requires both more detailed knowledge of pest species and their natural enemies and new technologies of culturing agents and monitoring their effects. Failure of past field trials can be attributed generally to lack of adequate information on the biologies of target and agent species.

To meet the requirements of biological alternative strategies, a greater and long term commitment to basic research is essential. With increasing concern for the environment and the need for alternatives to chemicals made useless because of resistance in pest species, the development of biological controls is receiving highest priorities. In order to promote biological and source reduction control in Manitoba, the province should consider the following key points: i.e., (i) a comprehensive prairie-wide survey of native predators and parasites, (ii) studies on pest mosquito species, emphasizing survival strategies for winter and drought conditions, and their interaction with native predators, parasites, and competitors, and (iii) provision of expertise on biological and source reduction strategies to all organizations in the province that are planning or conducting mosquito control programs.

References Cited

Angerilli, N.P.O. 1980. Influences of freshwater vegetation on the survival and oviposition by *Aedes aegypti* (Diptera: Culicidae). Can. Ent. 112: 1249-1252.

Asman, Sister M., P.T. McDonald and F.G. Zalom. 1980. Genetic manipulation of mosquitoes. Calif. Agric. 34: 23-24.

Bay, E.C. 1972. Biological control and its applicability to biting flies. pp 65-70 *in* A. Hudson (ed.), Symposium on biting fly control and environmental quality. Defence Research Board, Ottawa.

Bay, E.C. 1974. Predator-prey relationship among aquatic insects. Ann. Rev. Ent. 19: 441-454.

Chapman, H.C. 1974a. Biological control of mosquito larvae. Ann. Rev. Ent. 19: 33-59.

Chapman, H.C. 1974b. Nematode and protozoan parasites of mosquitoes and their potential use for control. pp 195-206 *in* Aubin, A., J-P. Bourassa, S. Belloncik, M. Pellissier, and E. Lacoursière. Le contrôl des moustiques/Mosquito Control, Univ. Quebec Press, Montreal.

Cress, F.C. 1980. Other mosquito predators. Calif. Agric. 34: 20

Ebeling, W. 1978. Pests attacking man and his pets. pp 440-444, *in* Urban Entomology. Univ. Calif. Press, Berkeley.

Ellis, R.A. 1980a. Fathead minnow, feasibility study, 1979 update. Progress rpt (unpublished). City of Winnipeg Insect Control Branch. 7 pp.

Ellis, R.A. 1980b. Annual Report (unpublished), City of Winnipeg Insect Control Branch. 19 pp.

Frederici, B. 1974. Virus pathogens of mosquitoes and their potential use in mosquito control. pp 93-136, *in* Aubin, A., J-P Bourassa, S. Belloncik, M. Péllissier, and E. Lacoursière. Le contrôl des moustiques/Mosquito Control, Univ. Quebec Press, Montreal.

Frederici, B.A., J. Fetter-Lasko, G. Soares and P.W. Tsao. 1980. Fungi show promise in biological control. Calif. Agric. 34: 25-27.

Gall, G.A.E., J.J. Cech jr., R. Garcia, V.H. Resh, R.K. Washino. 1980. Mosquito fish - an established predator. Calif. Agric. 34: 21-22.

Galloway, T.D. 1975. Application of a mermithid nematode (*Reesimermis nielseni* Tsai and Grundmann)from Louisiana for mosquito control in Manitoba. Proc. Alberta Mosquito Abatement Symposium, Univ. Alberta, Edmonton. pp 191-205.

Garcia, R., B.A. Federici, I.M. Hall, M.S. Mulla, C.H. Schaefer. 1980. BTI - a potent new biological weapon. Calif. Agric. 34: 18-19.

George, L.A. 1978. The potential of a local planarian, *Dugesia tigrina* (Tricladida: Turbellaria) for the control of mosquitoes in Ontario. Proc. entomol. Soc. Ont. 109: 65-69.

Hocking, B. 1972. Environmental quality and biting fly control: Problems and Possibilities. pp 9-14, *in* A. Hudson (ed.), Symposium on biting fly control and environmental quality. Defence Research Board, Ottawa.

Kale, H.W. II. 1968. The relationship of purple martins to mosquito control. The Auk. 85: 654-661.

Laird, M. (ed.). 1981. Biocontrol of medical and veterinary pests. Prager Publishers, N.Y. 235 pp.

Laird, M. (Chairman). 1982. Biting flies in Canada: health effects and economic consequences. NRCC No. 19248. 157 pp.

Mail, G.A. 1954. The mosquito fish, *Gambusia affinis*, in Alberta. Mosq. News 14: 120.

McHugh, K.A., L.S. Miller, and T.E. Oleson. 1968. Log pond mosquito control by larvivorous fish, pp 16-28 *in* The ecology and naturalistic control of log pond mosquitoes in the Pacific Northwest. Oregon State Bd Hlth.

Mulhern, T.D. (ed.). 1975. Manual for mosquito control personnel. California Mosq. Control Assoc. Press, Visalia, California. 384 pp.

Mulla, M.S., G. Majori, and A.A. Arata. 1979. Impact of biological and chemical mosquito control agents on nontarget biota in aquatic ecosystems. Residue Reviews 71: 121-173.

Rai, K.S. 1972. Genetic control of biting flies: progress and prospects. pp 79-88, *in* A. Hudson, Symposium on biting fly control and environmental quality. Defence Research Board, Ottawa.

Regan, D.G. and G.E. Rosberg. 1980. Investigations into the use of three-spined strickleback *(Gasterosteus aculeatus)* as a mosquito control agent for use in the Lower Fraser Valley. Beak Consultants unpubl. rpt. to Lower Mainland Regional District Mosquito Control Board, B.C. 24 pp.

Roberts, D.W. 1974. Fungal parasites of mosquitoes. pp 143-194, *in* Aubin, A., J-P Bourassa, S. Belloncik, M. Pellessier, and E. Lacoursière. Le Contrôle des Moustiques/Mosquito Control. Univ. Quebec Press, Montreal.

Shemanchuk, J.A. 1965. On the hibernation of *Culex tarsalis* Coquillett. *Culiseta inornata* Williston, and *Anopheles earlei* Vargas (Diptera: Culicidae) in Alberta. Mosq. News 25: 456-462.

Shemanchuk, J.A. 1977. Biological control of mosquitoes. Can. Agric. 22(4): 25-26.

Taylor, B.W., T.A. Harlos, and R.B. Brust. 1980. Coelomomyces infection of the adult mosquitoes *Aedes trivittatus* (Coquillett) in Manitoba. Can. J. Zool. 58: 1215-1219.

Trpis, M., W.O. Haufe, and J.A. Shemanchuk. 1968. Mermithid parasites of the mosquito *Aedes vexans* Meigen in British Columbia. Can. J. Zool. 46: 1076-1079.

Wraight, S.P., D. Malloy, and P. McCoy. 1982. A comparison of laboratory and field tests of *Bacillus sphaericus* strain 1593 and *Bacillus thiringiensis* var. *israelensis* against *Aedes stimulans* larvae (Diptera: Culicidae). Can. Ent. 114: 55-61.

Mosquito Abatement Programs in Manitoba

K. W. Plews, B.S.A. MSc.
Agrologist, Manitoba Environmental Management Division,
Environmental Control Services,
Box 7, Building 2 - 139 Tuxedo Avenue,
Winnipeg, Manitoba.
R3N OH6

Abstract

The legislation under which mosquito control in Manitoba may be carried out is explained. A list of 25 jurisdictions which had authority to carry out mosquito control in 1981 is given.

The following report outlines the registration procedure for the use of pesticides in Manitoba as described in The Manitoba Clean Environment Act. Section 14(1) of this Act basically states that any person intending to construct, alter, or set into operation any industry, undertaking, plant or process that will or may discharge contaminants into the environment is required to first file a proposal with Manitoba Environmental Management, unless exempted by Section 14.1 of the regulations. A proposal submitted under 14(1) sets into motion a department review and the Clean Environment Commission process.

Section 14.1 of the regulations serves as an exemption in that, where there will be no demonstrable effects beyond the boundary of a municipality, a municipal corporation may opt to file an impact statement, outlining the program and its possible environmental effects within and beyond the municipality with the Manitoba Environmental Management Division. Where this option is chosen, the proponent need not file a proposal under 14(1) of the Act.

Manitoba Regulation 156/74, a regulation under The Clean Environment Act dealing with pesticides, provides for the application of pesticides without submitting a proposal under Section 14(1) and 14(4) of The Clean Environment Act provided that the following conditions are met: (a) the application of the pesticide complies with the requirements of the Pest Control Products Act of Canada and the regulations thereunder, The Pesticides and Fertilizers Control Act and the regulations thereunder, and the use of recommendations prepared by the Manitoba Department of Agriculture; and (b) the person is an agricultural producer or a household applying on property owned or under his control, or on behalf of a Government Department, Crown Corporation or Municipal Corporation either to conform to the Noxious Weeds Act or to apply insecticide on an area not designated as residential or recreational.

Under this regulation, annual registration is required by Government Departments, Crown Corporations or Municipal Corporations prior to their conducting a spray program and by persons intending to spray over a body of water not wholly contained on their property.

It also states that, if the Minister of Health declares a health emergency, the regulatory requirements may be waived by the Minister responsible for the environment.

By implication of the above, all mosquito control applications by Crown Corporations, Government Departments or Municipal Corporations on lands designated as residential or recreational must comply with either Section 14(1) or 14.1 of The Clean Environment Act.

A list of Municipalities and Crown Corporations involved in mosquito control in 1981 is given below (Table I). Not all of those listed necessarily carried out a mosquito control program.

Table I. Municipalities and Crown Corporations Involved in Mosquito Control in 1981.

Jurisdiction	Adulticiding	Larviciding	Insecticide(s)
Birtle, Town of	x		Malathion
Brandon, City of	x	x	Malathion-Baygon-Abate-Flit MLO
Churchill, L.G.D. of	x		Malathion-Baygon
*Dominion City	x		Malathion
Emerson, Town of	x		Malathion
Flin Flon, City of	x		Malathion
Gillam, L.G.D. of	x	x	Baygon-Abate
Hartney, Town of	x		Malathion
Lac du Bonnet, Village of	x		Baygon
*Letellier, Town of	x		Malathion (Aerial)
Loni Beach		x	Abate
Man. Hydro: Grand Rapids	x		Methoxychlor
Jenpeg	x		Methoxychlor
Long Spruce	x		Methoxychlor
Melita, Town of	x		Air-Ground, Malathion
Morden, Town of	x		Baygon
Morris, Town of	x		Malathion
Bird's Hill Prov. Park	x		Malathion
Pinawa, L.G.D., of	x	x	Malathion-Abate
Portage la Prairie	x	x	Methoxchlor-Abate
*Steinbach	x		Malathion (Aerial)
Thompson, City of	x	x	Methoxchlor-Pyrethrins Abate-Dursban
*West St. Paul, R.M. of	x		Methoxchlor
*Winnipeg, City of	x	x	Various
Winnipegosis	x		Malathion

*Section 14.1 of C.E.C. Act

Emergency Mosquito Vector Control
by the City of Winnipeg

R.A. Ellis, B.Sc., M.Sc., Ph.D. Entomology, R.P.B.
City of Winnipeg Parks & Recreation Department
Insect Control Branch, 2799 Roblin Boulevard
Winnipeg, Manitoba R3R 0B8

Abstract

Coincident and coordinated with the Province's aerial spray program, the City of Winnipeg carried out an emergency mosquito adulticiding program between July 30th and August 11th, 1982. Emergency mosquito larviciding, from the ground and air, was also carried out in Winnipeg and 16 other surrounding municipalities between July 30th and August 26th, 1982. The cost of the City of Winnipeg's emergency program, subsequently recovered from the Province of Manitoba, was $211,447.15.

Immediately following the July 30th, 1982, meeting of the Manitoba Arbovirus Surveillance Committee (MASC) which confirmed that there was an emminent threat of Western equine encephalitis (WEE) to the people of southern Manitoba, the City of Winnipeg stepped up its mosquito control efforts, including both mosquito adulticiding and larviciding. When the health emergency was declared by the Province the following day, the City's Insect Control Branch had already raised its program from one of nuisance mosquito control to mosquito vector control.

During the relatively warm and calm evening weather that prevailed during early August, the activity of Culex tarsalis and the other potential vectors of WEE virus was high. To reduce the populations of these mosquito vectors, the Insect Control Branch used 10 fogging units whenever night-time weather conditions were favorable, including 3 thermal foggers to apply a methoxychlor in diesel oil solution and 7 cold foggers to apply either malathion on warm nights or propoxur on cool nights. Both aerial spraying, using a DC-6B, and residential fogging, using truck-mounted foggers, were carried out during the August 4th to 10th period.

The 2 adulticiding programs were coordinated so that fogging was carried out in a given area of the City of Winnipeg only until the aerial spray operations were completed in that same area. The City's fogging program could be considered a vital, stop-gap measure, designed to afford a degree of protection to as many residents as possible until the Province's aerial adulticiding program could be completed over Winnipeg.

The combination of ground and aerial adulticiding appeared to result in a dramatic decline in the numbers of mosquito vectors by mid-August.

Considerable effort went into the emergency larviciding program carried out by the Insect Control Branch. For nearly 4 weeks, between July 30th and August 26th, larviciding was carried out on a daily basis, working extended shifts. Portable and truck and helicopter-mounted sprayers were used to apply mosquito larvicides and pupicides. Dursban was the main larvicide that was used. Both granular and liquid formulations were applied to mosquito breeding sites. A Bell 47G4A helicopter was used to treat large, known breeding sites from the air. This helicopter is a very efficient tool for treating wide, highway ditches, rail and transmission line rights-of-way, and other inaccessible, off-road sites with Dursban 2.5G granules. Truck-mounted sprayers used Dursban 2E liquid sprays to treat the narrower ditches, effectively killing both mosquito larvae in pools and resting adult mosquitoes in vegetation. Some residual spraying with this insecticide was carried out in mosquito harbouring sites, including shrub and flower beds in recreational areas. Flit MLO was used to destroy mosquito pupae whenever these were found in breeding sites. Larviciding included the area within and up to 24 km beyond the City of Winnipeg, nearly 4500 sq. km.

The 17 municipalities included in the larviciding program, in whole or in part, included the following: i.e., Winnipeg, Selkirk, Stonewall, Stony Mountain, Grosse Isle, Rosser, St. Francois Xavier, Springstein, Oak Bluff, LaSalle, Grande Prairie, Ile des Chenes, Lorette, Dugald, Oakbank, Bird's Hill, and Lockport. This was the first time that the Insect Control Branch had carried out such an extensive mosquito larviciding program. Not only did it provide some protection to the residents of all these communities but also it demonstrated the feasibility of a regional mosquito abatement district encompassing this high population density area.

The total cost of the City of Winnipeg's emergency mosquito vector control program was $211,447.15, including both larviciding and adulticiding. These emergency costs, representing about 10% of the overall costs of the health emergency, were later recovered from the Province of Manitoba.

Acknowledgements

The author acknowledges the support and encouragement provided by Mr. D.G. Henderson, Commissioner of Environment and Mr. C.R. Burkett, Operations Supervisor, and Miss J.L. Buth, then Research Supervisor of the Insect Control Branch. All the staff members of the Insect Control Branch are to be congratulated for their dedication to their duties during the health emergency. Without these people, there would undoubtedly have been many more victims of WEE in 1981.

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

The Role of the Manitoba Emergency Measures Organization in the 1981 Health Emergency

N.W. Reilander

Supervisor Planning & Research
Manitoba Emergency Measures Organization
15th Floor, 405 Broadway Avenue
Winnipeg, Manitoba R3C 3L6

Abstract

A brief description of the role of the Manitoba Emergency Measures Organization during the 1981 health emergency is given, including interactions with the suppliers of equipment and materials, operations staff, the public and various provincial committees and agencies.

The surveillance program carried out by the Manitoba Arbovirus Surveillance Committee during the spring and summer months of 1981 indicated a high risk of Western Equine Encephalitis (WEE) in southern Manitoba. This was reported to the Deputy Minister of Health who, on 24 July 1981, notified the Manitoba Emergency Measures Organization (EMO) about the potential health emergency.

In accordance with its assigned responsibilities, EMO alerted other involved provincial and federal departments and, on 27 July 1981, chaired the first meeting of the Central Task Team to review and evaluate the WEE threat. The Central Task Team reported and made recommendations to the Deputy Minister Committee which in turn briefed the Cabinet Committee. As a result the Minister of Health was authorized to take any necessary action, including the implementation of an aerial spraying program.

On 28 July 1981, EMO made preliminary arrangements for aerial spraying by placing the aerial applicator on standby and procuring one-half of the estimated requirement of the insecticide Baygon MOS. With the assistance of Emergency Planning Canada, EMO arranged for the necessary Transport Canada authority for low altitude flying, for the establishment of an aerial spraying operations site at the Winnipeg International Airport (WIA) and for the provision of special weather forecasts by the Atmospheric Environment Service.

Following the Minister of Health's declaration of a Health Emergency on 30 July 1981, EMO confirmed arrangements with Conair Aviation Limited for the DC 6B aircraft and with Chemagro Limited for the provision of Baygon MOS. The EMO operations vehicle and trailer were deployed to the operations site at WIA, radio and telephone communications were established and support arrangements made for the Conair aircraft and crew, Chemagro personnel and the on-site operations staff.

At the same time a Provincial Information Centre was established by EMO in its offices and staffed on a daily basis from 8:00 a.m. to midnight by an EMO Supervisor and representatives from the provincial departments of Health, Agriculture and the Environment. The Provincial Information Centre, along with a News Centre set up by the Manitoba Information Services Branch, were the primary means of dissemination information about spraying operations to the general public. In addition, the Provincial Information Centre was able to respond to individual citizen inquiries from all parts of the province. Special telephone lines were provided to ensure a rapid and coordinated flow of information between the operations site at WIA, Information Services, the Provincial Information Centre and EMO.

Because the pace of the operation did not require the activation of the Provincial Emergency Coordination Centre, coordination of the spray operations was provided by the EMO staff from its regular office accommodation. The overall control and direction of the operation was provided by the Central Task Team which met almost daily to review the WEE threat and to make decisions and/or recommendations to the Deputy Minister Committee. EMO through its municipal advisors, provided direct contact with the local authorities of the communities to be sprayed and kept them informed about the spraying program.

The aerial spraying operations began on 4 August 1981 with the spraying of West Winnipeg. From this point on, the emergency response settled into a routine of almost daily meetings of the Central Task Team and the Deputy Minister Committee at which the WEE threat was reviewed, the risk assessed and decisions taken with respect to areas to be sprayed. Thereafter, the WIA on-site operations staff planned the detail of missions to be flown and passed this information to Information Services (for the media), the Provincial Information Centre (for the public) and to EMO (for municipal and other authorities).

Upon completion of the spraying operations, on 26 August 1981, EMO closed down the Provincial Information Centre after having logged a total of 7,720 telephone calls from throughout the southern part of the province. EMO closed down the WIA operations site on 27 August 1981, following the completion of 25 spraying missions which covered 4 cities and 17 towns. The health emergency was officially declared terminated on 24 September 1981. EMO coordinated the preparation of a final provincial report on the emergency and the subsequent preparation of Emergency Action Guidelines for any future WEE health emergency.

Emergency Mosquito Vector Control Operations

R.A. Ellis, B.Sc., M.Sc., Ph.D. Entomology, R.P.E.

City of Winnipeg Parks and Recreation Department
Insect Control Branch, 2799 Roblin Boulevard
Winnipeg, Manitoba R3R 0B8

Abstract

A descriptive overview of the Province of Manitoba's 1981 emergency mosquito control operations is given including the key considerations in selecting spray equipment and materials, criteria for spraying and summary program statistics.

Vector control during the most recent Western Equine Encephalitis (WEE) outbreak was accomplished because some advance warning and preparation was possible. The Manitoba Arbovirus Surveillance Committee (MASC) currently provides a 1-2 week advance warning of an incipient outbreak. This gives the Province adequate time to assemble and implement an emergency aerial spray program designed to break the *Culex tarsalis*/WEE virus transmission cycle in high-risk areas. Most of the people at risk in Southern Manitoba live in urban centres. By reducing the numbers of mosquito vectors present in such areas, the risk that people will be bitten by virus-carrying mosquitoes is decreased proportionately.

The risk of WEE to Manitobans was perceived to be high by MASC in mid-July, in 1981. Mosquito control remains the only practical approach to minimizing WEE virus transmission to people. During a health emergency, mosquito control must be limited to an efficient insecticide spray program which is aimed at destroying adult female *C. tarsalis*, a percentage of which carry WEE virus from wild hosts to horses and humans. Under normal circumstances, a fully integrated approach to mosquito control would be preferred. Such a program would include the aerial and ground application of insecticides to control larval and adult nuisance mosquitoes, the reduction of mosquito breeding sites, and extensive research and public education programs. When many widely-separated communities at risk must be protected as quickly as possible, the aerial application of mosquito adulticides is considered to be the most appropriate method to reduce the number of potential vectors (Anon., 1978; Breeland *et al*., 1980).

Large, multi-engine aircraft, outfitted with the required ultralow volume (ULV) spray system, can treat an average small community (165 km^2) within 1 hour, both efficiently and safely. In 1981, as during previous outbreaks, a DC-6B was used. This 4-engine, converted passenger aircraft has all the modern safety features essential for low-level flights over urban centres. In addition, it has sufficient insecticide carrying capacity to treat an entire urban centre, including buffer zone, during 1 flight. One properly-equipped DC-6B is equivalent to 70 conventional agricultural aircraft but the problems and costs associated with coordinating and maintaining such a large fleet of small planes is avoided.

Conair Aviation Limited of Abbotsford, B.C., was chosen as the aerial applicator. This firm not only uses the best available spray aircraft, spray system and guidance technology and employs well-trained, experienced and responsible pilots, but also is dedicated to public health protection in its many types of aerial service programs.

The material applied for vector control is just as important as the mode of application. The choice of insecticide for mosquito control in an aerial ULV application in 1981 was limited to 2 materials: i.e., malathion and propoxur. The 2 key criteria in deciding between the 2 materials are safety and effectiveness. Although known to be both safe and effective, based on use-experience in numerous major vector control programs in many countries (e.g., Gardner and Iversen; 1974; Mitchell *et al*., 1969, 1970), malathion is believed to lose its effectiveness against mosquitoes at temperatures below 18°C. However, this limitation is not documented in the scientific literature and research in this regard is needed. In any case, because of this suspected limitation and because early morning and late evening temperatures often fall below 18°C in southern Manitoba during August, the main period of WEE risk, this insecticide had to be rejected.

Thus, propoxur was the only alternative available to the Province. Based on provincial experiences in 1975 and 1977, propoxur has been shown to be effective against mosquitoes and without significant adverse effects on the environment (Brust and Ellis, 1976; Bowen *et al*., 1976). Baygon MOS, or "mosquito oil solution", is a 12% formulation of propoxur dissolved in solvent and oil and produced by Chemagro Ltd. It has been widely used in Canada for mosquito control by federal, provincial, municipal and private agencies since the mid-70's and is now gaining popularity in many mosquito abatement jurisdictions in the U.S.A.

Baygon MOS may be applied at a rate of 275 to 1100 ml/ha, according to federally-registered label recommendations. In 1981, as in previous outbreaks, Baygon was applied at 438 ml/ha because this is the lowest rate at which the material can be applied under Manitoba conditions to obtain an acceptable level of reduction in populations of *C. tarsalis*. This application rate is equivalent to 6 U.S. fl. oz./acre or 52.5 g active ingredient/ha (or 0.75 oz. a.i./a). This relatively low rate of application attests to the high activity that propoxur has against adult mosquitoes.

As soon as an incipient outbreak was recognized, the probable aerial applicator and insecticide manufacturer were alerted and relevant contingency plans were updated. On July 30th, 1981, the Minister of Health declared a provincial health emergency due to WEE. The Manitoba Emergency Measures Organization (EMO) then began to coordinate the supply of staff, equipment and materials for the aerial spray operations. EMO's attention to a host of operational details was essential to the success of the spray operations that ensued.

The emergency spray operations were based out of the Winnipeg International Airport. EMO supplied and positioned the truck-mounted field office and the trailer. The office was equipped with tables, chairs, shelves, cabinets, lights, telephones, 2-way radio communications and a telecopier. The trailer served as a meeting-lunchroom. All spray flights were flown from this site.

Aircraft loading was handled as efficiently and safely as possible to make the best use of those brief periods when spray conditions were optimum for mosquito control. From the storage tanker(s), the insecticide was drawn through 7.6 cm hoses by 2 tandem trash pumps at a rate of about 750 l/min. When the tanks inside the fuselage of the DC-6B were nearly filled, first one, then the other, pump was shut down. Quick couplers at all connection points minimized leaks and spills when hoses were moved. An in-line meter accurately measured the volume of each load. A high-volume filtering system, on the ground and in the aircraft, minimized the risk of contaminants plugging the special nozzles used. Using the tank configuration in place during 1981, the DC-6B had a maximum insecticide capacity of about 13500 l. In flight, an on-board pump would move the insecticide through Conair's flow control and filtering system to the above-wing booms. Each boom supported 8 knife-edged uprights on which Beecomist 360 atomizers were mounted. An electrically-driven motor spun the stainless steel, 40 micron sleeve of the atomizer at about 12,000 rpm, producing Baygon droplets with diameters, upon emission, of about 40 microns.

In flight, all aspects of the ULV application were carefully controlled from the cockpit, including the flow control system (FCS) and guidance over the spray block. Conair's FCS enables the insecticide flow rate to be varied automatically with changes in aircraft speed. The Litton LTN-51 guidance system provides precise navigation to and over the spray block. While spraying, the DC-6B flew at 60 or 90 m (using dual altimeters) and at a groundspeed of 185-200 knots.

The criteria for aerial spraying included biological, meteorological and operational considerations. The 2 main biological factors considered were the activity periods of mosquitoes and honeybees with the objects of maximizing mosquito kill and minimizing bee kill. Because spraying could only be done under daylight conditions, flights were made in the very early morning and in the evening.

Weather conditions were another major consideration. Temperature, relative humidity, windspeed and wind direction and the presence or absence of a temperature inversion below the altitude of the DC-6 while spraying were all important factors. Generally, spraying was done during warm, humid periods when winds were light and there was no pronounced temperature inversion.

However, a DC-6, to some extent, makes its own weather. The influence of an aircraft's wake on the downwind dispersal of ULV sprays should not be underestimated (Boyle et al., 1974). The entire vortex system sinks towards the ground with an average downward velocity of about 0.7 m/sec. The descent of the vortex system containing the spray drops stops when the centers of the wing-tip vortices are about 15 m above the ground. The effects of crosswind and turbulence are probably only important after most of the droplets have dropped in a spinning motion to a level where they will begin to impinge on flying mosquitoes.

The operational criteria included the locations to be sprayed, as determined by the Cabinet Task Force, the ready supply of insecticide, the availability of the aircraft and the proper functioning of all the equipment, both on-board the aircraft and on the ground. And, as mentioned above, only daylight flights were permitted, for insurance and safety reasons.

Assuming that all the criteria had been met, the aircraft would be fueled and readied for take-off. The proper coordinates for the location to be sprayed would be entered into the on-board computer and the DC-6 would depart for the spray block.

In deciding to spray a given community, the Province was guided by the principle of protecting the largest centres of population known to be at risk first and then moving to the smaller centres at risk. Most of the communities involved in the emergency mosquito control operations (Table 1) were centered in spray blocks measuring 13.7 x 12.9 km. Within each spray block, 17 flight lines, running north-south, were flown at 0.8 km intervals. The spray blocks were designed in this way to effectively control the mosquitoes in the centre of the spray blocks, where most of the people lived, and to provide a 4 to 6 km buffer zone around the built-up area in order to reduce the chances that mosquitoes in this area would disperse into the town.

The summary statistics for the 1981 emergency aerial spray operations follow: i.e., 455,545 ha were sprayed; 199,478 l of Baygon MOS were applied; and $1,977,030 were spent on direct costs (including $550,762 for the DC-6B and $1,426,268 for the Baygon (MOS). In terms of per capita costs, the program cost about $2.60 per person protected.

Acknowledgements

As Technical Officer in charge of Field Operations while seconded to the Province of Manitoba, I was ably assisted by persons expert in the areas of mosquito biology and control. Valuable advice and assistance was provided by Dr. G. Surgeoner, University of Guelph; B. Madu and B. Robinson, Conair Aviation Ltd.; R. Misener, Chemagro Ltd.; J.L. Buth, R.L. Cochrane, F.S. Richards and R. Burkett, City of Winnipeg Parks and Recreation; and H. Eckert and staff, Manitoba Emergency Measures Organization. The efficient and successful completion of the mosquito control program was due to the advice given and the long, difficult hours worked by these key people.

Table 1 - Emergency mosquito control operations over 21 communities in southern Manitoba during August, 1981.

August Date/Time	Location	Baygon MOS (L)	Area (ha)
4th/a.m.	Winnipeg [1]		
7th/a.m.	Winnipeg		
7th/p.m.	Winnipeg		
10th/p.m.	Winnipeg		
11th/a.m.	Winnipeg	43229.3	98599
11th/a.m.	Portage la Prairie	7722.2	17611
11th/a.m.	Selkirk	7722.2	17611
11th/p.m.	Morris	7729.4	17611
12th/p.m.	Steinbach	7724.9	17611
12th/p.m.	Stonewall	7722.2	17611
13th/a.m.	N. Morden/ Winkler	7735.5	17611
13th/p.m.	S. Morden/ Winkler	7734.9	16923
14th/a.m.	Brandon	9396.1	21403
14th/p.m.	Virden	6591.9	15363
18th/a.m.	Dauphin	7735.5	17611
19th/a.m.	Gimli	10188.4	23243
19th/a.m.	Altona	7724.9	17611
20th/a.m.	Beausejour	7726.4	17611
20th/a.m.	Carman	7722.2	17611
20th/p.m.	Neepawa	7727.9	17611
21st/p.m.	Minnedosa	7726.4	17611
24th/p.m.	Russell	7722.2	17611
25th/p.m.	Swan River	7717.7	17611
25th/p.m.	Killarney	7722.2	17611
26th/a.m.	Roblin	6950.4	15851
		199,672.4	455,547

---

[1] Four flights were required to treat the City of Winnipeg.

References Cited

Anon., 1978. Control of Western Equine Encephalitis. U.S. Hlth, Educ. and Welfare, Publ. Hlth Service, CDC, Vector Topics No. 3, 33 p.

Boyle, D.G. et al., 1974. DC7B aircraft spray system for large-area insect control. U.S. Army Report DGP-DR-C980A, 203 p.

Bowen, W.G. et al., 1976. Environmental monitoring of insecticide applications during the emergency mosquito control operation in Manitoba, 1975. Can. J. Publ. Hlth (Special Suppl. Western Encephalomyelitis) 67:63:68.

Breeland, S.G. et al., 1980. Vector Control p. 605-622 In T.P. Monath and W.C. Reeves (Eds.) St. Louis Encephalitis. Amer. Public Hlth Assoc., Wash., D.C. 680 pp.

Brust, R.A. and R.A. Ellis, 1976. Assessment of the emergency mosquito control operation in Manitoba, 1975. Can. J. Publ. Hlth (Special Suppl. Western Encephalomyelitis) 67:69-71.

Gardner, A.L. and R.E. Iverson, 1968. The effect of aerially applied malathion on an urban population. Arch. Environ. Health 16:823-826.

Mitchell, C.J. et al., 1969. Effects of ultralow volume applications of malathion in the Hale County, Texas. I. Western encephalitis virus activity in treated and untreated towns. J. Med. Ent. 6:155-162.

Mitchell, C.J. et al., 1970. Effects of ultralow volume application of malathion in Hale County, Texas. II. Mosquito populations in treated and untreated areas. J. Med. Ent. 7:85-91.

Drift & Deposition During Aerial Spraying of Baygon over Winnipeg August 1981

G. Barrie Atkinson

Atmospheric Environment Service
Environment Canada
1000 - 266 Graham Avenue
Winnipeg, Manitoba

Abstract

The forces which act upon small droplets in the atmosphere are delineated. Those forces which are considered significant are then discussed with respect to observations made during the spraying program in August 1981.

Large particles in the atmosphere, such as droplets of 100 µm or greater are not subject to appreciable drift, except in very strong winds. Those with diameters between 20 and 100 µm, although carried along by the wind, have significant terminal velocities. They are therefore dispersed as plumes that fall. Still finer droplets are more influenced by eddies than by gravity, and are transported greater distances downwind. Average drift distance for droplets of various sizes are given (Table 1).

The monitoring by the Environmental Protection Service (EPS), Environment Canada of the Baygon sprayed over Winnipeg in August, 1981 shows droplets that are as small as about 5 µm. Droplets of this size should drift about 35 km in a 5 km/h wind while falling 10 m; or 320 km while falling from 90 m. This distance is the extreme, but a droplet with a diameter equal to the measured numerical median diameter (NMD) of less than 20 µm should drift at least 1.15 km in a 5 km/h wind while falling 10 m, or more than 10 km while falling from 90 m.

An attempt will be made in this paper to explain the physical processes which bring small droplets (in the range 4 to 40 µm diameter) to the surface from an altitude of 90 m within 1.5 h.

Non Meteorological Data

Aerial spraying was carried out in August 1981 over Winnipeg, Brandon, Portage La Prairie and a number of other smaller communities in Manitoba to control mosquitoes in an attempt to avoid an outbreak of Western Equine Encephalities. The aircraft used, application rates, and tracks flown are given by Ellis (1981).

EPS monitored the aerial spraying by setting out a series of teflon-coated slides at 3 locations (Fig. 1) in the vicinity of Winnipeg (A, B, C), two locations near Portage La Prairie (D, E) and at two other Manitoba towns (Youmans, 1981). Only the slides collected at Winnipeg and Portage La Prairie were counted.

The frequency count sheets were obtained from EPS and entered into the Atmospheric Environment Service (AES) regional computer. A program was written to calculate the NMD of each slide or groups of slides. The results of some of these calculations are shown (Table 2).

A statistical assessment was made of the differences noted in the average number of droplets on slides which were left exposed for different times (Steel and Torrie, 1960, pp. 67-87). It was found that there was no significant difference between slides exposed for a nominal half an hour and those exposed for a nominal hour. There was however, significant difference between slides exposed for a nominal hour and a half and those exposed for a nominal one hour; and a highly significant difference between those exposed for a nominal hour and a half and those exposed for a nominal half an hour (Table 3). Since the average number of droplets increased with time, this indicates that the slides were not exposed long enough. Presumably, with longer exposure, a higher proportion of smaller droplets would have reached the slides and the NMD would have decreased.

External Forces

The externl forces known to act on a free moving droplet include (Goering et al, 1972):

a. Van der Wall's forces - these forces arise from the distortion of the charge distribution within a droplet which is subjected to the field of another droplet.

b. Thermophoresis - a force which occurs along a temperature gradient due to unequal molecular bombardment.

c. Photophoresis - which is the tendency of droplets to migrate when illuminated by a beam of light.

d. Diffusiophoresis - a force which occurs along a concentration gradient in the direction of the diffusion flux of the heavier gas component.

e. The Basset effect - the diffusion of the vorticity away from the turbulence in the wake of the droplet (important only during periods of acceleration).

f. Buoyancy.

g. Gravity.

h. Drag.

i. External electrical gradient.

j. External pressure gradient.

The first five of these forces (a to e) have been shown by a number of investigators to be negligible under normal atmospheric conditions if the droplets are large enough; that is, if the droplets are above the submicron range.

An electric charge on droplets (i) can significantly alter their movement because of the electromagnetic field of the earth and the electrostatic field which is normally present (about + 100 Vm^{-1}) near the surface of the earth (Golde, 1977, p. 501). In this case, no attempt was made to place a charge on the droplets as they left the spray nozzle. However, it is very likely that the static charge that usually accompanies rapidly moving fluids was present to some extent. No attempt was made to measure this change, and it will have to be ignored in this study.

Buoyance and gravity (f and g) may be handled together by the following equation (Goering et al, 1972):

$$W = g\left(\rho_l - \rho_a\right)\frac{\pi D^3}{6} \tag{1}$$

where W - weight of the droplet in air
ρ_l - density of the liquid in the droplet
ρ_a - density of air
D - diameter of the droplet
g - acceleration of gravity

The weight (W) is the gravity force minus the bouyance force.

The drag force on a droplet (h) is given by the equation (King et al, 1953, p. 305):

$$D_f = C_d \frac{\rho_a A V^2}{2} \tag{2}$$

where D_f - drag force
C_d - dimensionless drag coefficient
A - projected area of the particle = $\frac{\pi D^2}{4}$

V - velocity of the particle relative to the medium through which it is falling.

The method used by Berry and Pranger (1974) for calculating the terminal velocity of droplets in air at values of pressure and temperature found in the atmosphere includes the value of the drag force. They attributed the method to Davis (1945) who developed it to derive the terminal velocity of rigid spheres. This method consists of finding a regression relationship between the Reynolds number (Re) and C_dRe^2. The Reynolds number is the ratio of the inertial force to the viscous force (Hess, 1959, p. 273) and can be directly related to the size of the droplet.

Since $\mathrm{Re} = \dfrac{DV\rho_a}{\eta}$ $$(3)$$

where η is the dynamic viscosity of the air

and $$C_d = \frac{2\,mg}{\rho_a A V^2} \qquad (4)$$

where m is the mass of the drop

then the product $$C_d \mathrm{Re}^2 = \frac{8\,mg\,\rho_a}{\pi\,\eta} \qquad (5)$$

is independent of the velocity V and the least squares fit approach is justified.

Berry and Pranger actually developed a number of different regression equations, fitting the data of different researchers and different ranges of Reynolds number. Reid and Crabbe (1980) in their study of long range drift of aerial spray in New Brunswick forestry chose the following equation as being most appropriate:

$$\mathrm{Re} = a_1X + a_2X^2 + a_3X^3 + a_4X^4 \qquad (6)$$

where $X = C_d\mathrm{Re}^2$

and
$a_1 = 0.412657 \times 10^{-1}$
$a_2 = -0.150074 \times 10^{-3}$
$a_3 = 0.758804 \times 10^{-6}$
$a_4 = -0.168841 \times 10^{-8}$

The terminal velocity is then calculated from equation 3.

The external pressure gradient (j) is nothing more than the wind, which for the locations of interest must be estimated from the nearest meteorological observation site; Winnipeg International Airport, or Canadian Forces Base Southport, near Portage La Prairie. The surface wind at these locations is measured at the World Meteorological Organization standard height of 10 m.

Winds at other heights above ground must be estimated from more distant stations, and from observations taken at times more remote from the time of interest. The best estimates of the 10 m and 90 m winds at the monitoring locations derived from aerological data is given (Table 4).

The wind is not constant with height, but may be approximated by a power law of the form: (Hess, 1959, p. 278)

$$\frac{u_z}{u_o} = \left(\frac{z}{z_o}\right)^p \qquad (7)$$

where u_o - one minute mean wind at observational height
u_z - one minute mean wind at height z
z - height above ground of interest
z_o - height at which u_o is measured

and the power p varies with the stability of the atmosphere. A study by Fraser and Bellan (1969) conducted at a site west of Stonewall, Manitoba developed the dependance of p on time of day and windspeed:

$$p = (.325 - .009\,u_{10}) \left[\cos\left[2\pi \left(\frac{T-1}{24} \right) \right] + 1 \right] + 0.09 \qquad (8)$$

where T is the local standard time on a 24 hour clock and u_{10} is the 10 m mean wind speed for 1 minute in miles per hour. The value of p calculated is given (Table 4).

The direction of the wind usually varies with height according to a clockwise (in the Northern hemisphere) spiral named after W. F. Ekman (Hess, 1959, p. 281).

The equations for the Ekman spiral are:

$$u = u_g(1 - e^{-az} \cos(az)) \quad \text{and} \quad v = u_g e^{-az} \sin(az) \qquad (9)$$

where u - eastward component of the wind
v - northward component of the wind
u_g - eastward component of the geostrophic wind, which is the wind above the frictional effects of the earth's surface

and $a = \sqrt{f/2K}$ $(\simeq 10^{-5}\ m^{-1})$
where f - Coriolis parameter ($\simeq 10^{-4}\ s^{-1}$ at mid latitudes)
and K - eddy viscosity coefficient ($\simeq 5\ m^2\ s^{-1}$)

From the wind components u and v the wind direction in degrees from true north is calculated. The results of applying equations 7 and 9 are included, for representative levels, in Tables 7 to 10, and may be compared at the 90 m level in Table 4. On average, the 90 m wind calculated from the 10 m wind is the same as the estimated 90 m wind (11.5 kts). This indicates that using the calculated winds would not change, on average, the amount of drift.

Evaporation of the Droplet

It is assumed that the initial size distribution of the droplets is narrow and centered near 40 μm. Monitoring indicates that the NMD at the surface for all locations taken together is about 17 μm.

This implies that the diameter of the average droplet decreased by 23 µm, and its volume by more than 90% (Table 5). But coalescence occurs as well as evaporation, and the fewer larger drops which result from coalescence carry a very high percentage of the total volume to the surface. Since all drops greater than 66 µm were lumped into one category it was not possible to accurately estimate this effect.

Two attempts were made to quantify the rate of decrease of droplet diameter with time using theoretical equations, but neither proved successful. It was realized that the evaporation increases with decreasing diameter, so that the relationship $\frac{dD}{dt}$ is not linear. Both the theoretical equations examined (Fletcher, 1962; Goering et al, 1972) were developed for water droplets and required several parameters which proved impossible to find for the spray liquid. These included; the diffusivity of the spray vapor in air, the effective molecular weight of the spray liquid, the saturation wet bulb temperature of the spray liquid, the vapor pressure of the spray liquid, and the latent heat of vaporization of the spray liquid.

An emperical equation is given in Trayford and Welch (1977), for the evaporation of water-based droplets.

$$\frac{D}{D_o} = \left(1 - \frac{t}{G}\right)^{1/2} \tag{10}$$

where D_o - the initial droplet diameter (um),
t - the time (s)

and $G = \frac{Do^2}{H \Delta T}$

where ΔT is the wet bulb depression with respect to the spray liquid and H is derived from basic heat transfer properties.

The formula for G also includes the same parameters for which we could not find values before, such as; wet bulb depression of the spray liquid, the specific heat of the spray liquid, its dynamic viscosity, and its thermal conductivity. However we do have an estimate of D, the NMD; and t, the exposure time, and if D_o is assumed near 40 micrometers, then G may be calculated. This was done in the hope that a relatively constant G might be found. The disappointing results are shown in Table 2. Only for location E was the value of G approximately constant.

The known theoretical possibilities having been exhausted, evaporation from the droplets was treated in a heuristic method. The terminal velocity of a 40 μm droplet was calculated using equations 3 to 6, which then gave the time to fall one meter. The droplet was evaporated according to equation 10 for this time to give a new diameter, and another terminal velocity was calculated. This procedure was continued until the droplet was less than 2 μm in diameter. The method was repeated using different values of G until the time (about 70 minutes) and the droplet diameter (about 17 μm) were appropriate for a fall of 90 m. The value of G = 41.0 minutes gave the results in Table 6.

These figures are supported by the somewhat larger measured NMD at the two locations (D and E) near Portage La Prairie. It was uncertain from the references whether this community had been sprayed from an altitude of 60 m or 90 m. The calculated drop diameter of 27 μm after a fall of 60 m compared very well with the average NMD (29.4 μm) from these locations, and suggests that they were indeed sprayed from 60 m.

Discussion of Results

The results of combining the changing terminal velocity of the droplets due to evaporation, and the wind field as calculated from the power law and the Ekman spiral are summarized for each location in Tables 7 to 10. The calculations indicate that the "average" sized droplets are advected about 18.5 km at location A; 8.7 km at location B; 9.6 km at location C; and 9.9 km at locations D and E.

At location A the maximum possible upwind location of spraying was approximately 13 km; at location B 15 km; at C about 10 km; at D nearly 7 km and at location E about 2 km.

Thus, except for location B, the droplets appear to have been advected a distance which would place their origin outside the spray area.

Two possible causes of this discrepancy are known. The first is that the aircraft imparts downward momentum to the volume of air into which the liquid is sprayed, and that this causes an effective spray height which is much lower. This is believed to be the case. The downward thrust would depend upon the speed, weight, and physical shape of the aircraft.

The second is that what is observed on the ground does not arrive there due to gravity alone, but is carried downward in some areas (and upward in others) due to turbulence. The atmosphere, particularly the lowest levels, is rampant with eddys of all scales, both horizontal and vertical and all angles between. This implies that in some spots the spray droplets encounter a rising column of air and in others a downdraft. This is the mechanism through which one might expect spray paths half a mile apart to cover a target area with a more or less even distribution of chemical. If turbulence brought those droplets which were observed downward more rapidly than gravity alone, then the distance advected by the wind would be much less. However, it is important to point out that for every downdraft there would have to be a corresponding updraft; and it is reasonable to assume that just as many of the droplets are slowed in their fall (and advected further) as there are "down" drops (which are observed). Those which are slowed sufficiently simply evaporate completely.

Conclusion

With the assumptions that the droplet size at source is 40 μm, and that the NMD size is representative of those droplets which have been affected by turbulence in upward and downward directions equally, then an procedure for calculating the evaporation and terminal velocity of the droplets has been developed.

The estimates of the wind field are the best available, and better than most locations where on site wind measurements are not taken, because of the proximity of first order meteorological observing sites. However, this is not sufficient to define the mechanisms by which these very small droplets reach the ground within the spray area.

Acknowledgements

Thanks are due to: Roy Ellis and Terry Youmans for providing data used in this study; Bob Tortorelli for the preparation of diagrams and formulae, and to Patti Welwood for typing.

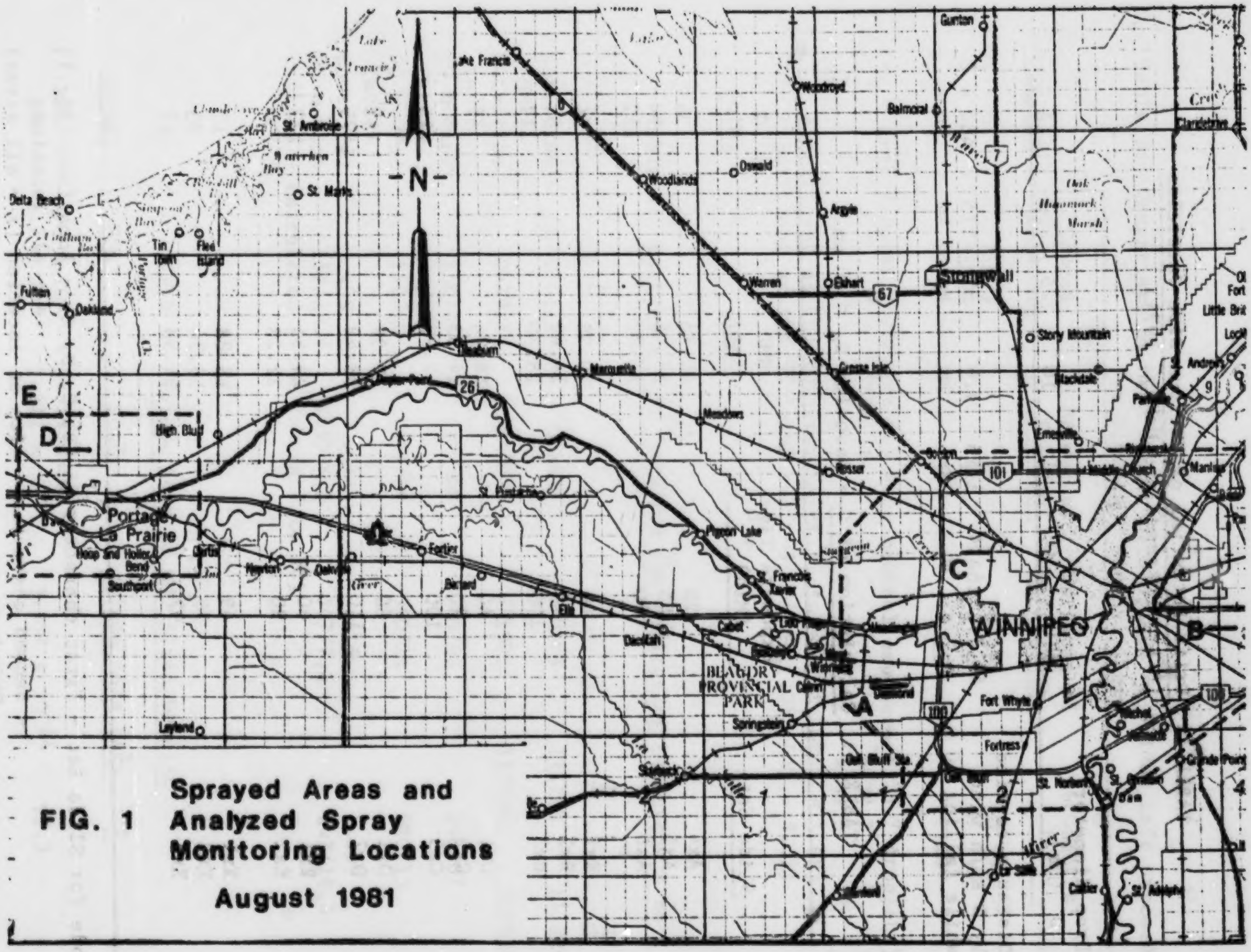

N
A
B
C
D
E
1
2
4
WINNIPEG
Portage La Prairie
Stonewall
Delta Beach
Fulton
Oakland
Tin Town
Flee Island
St. Ambroise
St. Marks
Lake Francis
Woodlands
Oswald
Woodroyd
Balmoral
Gunton
Argyle
Warren
Elshart
Grosse Isle
Marquette
Meadows
Rosser
Stony Mountain
Headingley
Pigeon Lake
St. Francois Xavier
Cabot
Dacotah
Elie
Fortier
Oakville
Newton
Southport
High Bluff
Springstein
Starbuck
Oak Bluff
Fort Whyte
Fortress
St. Norbert
Leyland
BEAUDRY PROVINCIAL PARK
Clandeboye
Lockport
Little Britain
St. Andrews
Middle Church
Grande Pointe
St. Adolphe
Oak Hammock Marsh
26
67
7
101
100
6
9

FIG. 1 Sprayed Areas and Analyzed Spray Monitoring Locations

August 1981

TABLE 1: Drift Potential of Various Size Particles
(After Mukammal and McKay, 1971)

Drop diameter D (µm)	100	50	20	10	5	2	1
Distance carried by 5 km/h wind while falling 10 m	49.7 m	184.4 m	1.15 km	4.6 km	35 km	115 km	460 km

TABLE 2: Calculation of G - Assuming $D_o = 40$ µm

Slide Set	Exposure Time (Min)	NMD (um)	G (Min)
Ax4	25	20.76	34
Ax5	55	17.45	68
Ax6	90	17.62	112
Bx1	40	20.58	54
Bx2	70	14.63	81
Bx3	100	15.20	117
Cx1	40	14.94	46
Cx2	70	14.35	80
Cx3	100	17.98	125
Dx1	80	27.27	149
Dx2	110	38.48	1475
Ex1	60	29.55	132
Ex2	90	22.28	130
Xx1	49	18.88	63
Xx2	79	16.34	95
Xx3	97	16.50	117

Code for Slide Set - First character (A, B, C, D, E) - locations (Fig.1)
x - all locations
- Second character - site at location (x = all sites)
- Third character - exposure time code

Table 3: Comparison of Mean Number of Droplets on Slides Left Exposed For Different Nominal Times

Hypothesis	Common Variance	Degree of Freedom	Variance of Difference	Student's t
$u_1 = u_2$	716.3	66	6.49	-0.693
$u_2 = u_3$	580.3	54	6.59	-1.73*
$u_1 = u_3$	695.3	54	7.21	-2.20*

(u_1 - mean droplet count for 0.5 hour nominal exposure = 23.7 droplets)
(u_2 - " " " " 1.0 " " " = 28.2 ")
(u_3 - " " " " 1.5 " " " = 39.6 ")
(* - significant at the 10% level - $t(.1, 54) \leq t(.1, 40) = 1.68$)
(**- significant at the 5% level - $t(.05, 54) \leq t(.05, 40) = 2.02$)

Table 4: Estimated Wind Data for the Monitoring Locations A to E

Location	A	B	C	D	E
Date/time (GMT)	050115	071420	080105	111200	111220
Date/time (LST)	041915	070820	071905	110600	110620
10 m wind (kts)	150/06	330/03	040/03	240/07	
90 m wind (kts)	150/10	340/10	050/11	280/15	
power (p)	.381	.334	.415	.439	
90 m wind (kts) - calculated (from Tables 7 to 10)	157/13.9	337/6.3	047/7.4	247/18.4	

TABLE 5: Volume Loss of NMD Sized Droplet

D(um)	D(m)	V(m^3)	Volume loss (m^3)	%
40	4.0×10^{-5}	3.351×10^{-14}		
17	1.7×10^{-5}	2.572×10^{-15}	3.094×10^{-14}	92.3

TABLE 6: **Calculation of Terminal Velocity, Drop Diameter and Time to Fall**

Assuming G = 41.0 minutes

D_o = 40 micrometers

Distance Fallen (m)	Terminal Velocity (ms^{-1})	Drop Diameter (um)	Time to Fall 1 m (s)	Cumulative Time (Min)
10	-0.0409	38.1	24.5	3.9
20	-0.0369	36.2	27.1	8.2
30	-0.0329	34.1	30.4	13.0
40	-0.0288	31.9	34.7	18.5
50	-0.0248	29.5	40.3	24.8
60	-0.0208	27.0	48.2	32.2
70	-0.0167	24.1	59.8	41.2
80	-0.0127	20.9	79.0	52.8
90	-0.00861	17.1	116.2	68.9
100	-0.00455	12.1	220.	96.
110	-0.00048	1.6	2070.	205.

Table 7
Location A

```
THE HOUR IS 19

THE WIND IS 150 6

THE SPRAY HEIGHT IS  90 METERS

HEIGHT   WIND    DISTANCE CUMULATIVE   DROP    TERMINAL  TIME FOR CUMULATIVE
       DIR   SP  ADVECTED  DISTANCE    SIZE    VELOCITY  1 M FALL    TIME
  M         M/S         M          M       M        M/S         S         S

   1  149.2 1.28    149.28     149.28 .1751E-04 -.861E-02    116.20   4136.33
  10  150.0 3.09    251.99    2229.35 .2090E-04 -.123E-01     81.58   3247.08
  20  150.9 4.02    246.61    4740.13 .2412E-04 -.163E-01     61.31   2532.00
  30  151.8 4.69    230.58    7119.73 .2695E-04 -.204E-01     49.12   1978.18
  40  152.6 5.24    214.67    9336.81 .2952E-04 -.244E-01     40.99   1526.00
  50  153.5 5.70    200.55   11404.28 .3189E-04 -.284E-01     35.17   1143.77
  60  154.4 6.11    188.30   13340.98 .3409E-04 -.325E-01     30.81    812.65
  70  155.2 6.48    177.69   15164.40 .3617E-04 -.365E-01     27.41    520.52
  80  156.0 6.82    168.44   16889.36 .3813E-04 -.405E-01     24.70    259.09
  90  156.9 7.13    160.31   18528.19 .4000E-04 -.445E-01     22.47     22.47
```

Table 8
Location B

```
THE HOUR IS  8

THE WIND IS 330 3

THE SPRAY HEIGHT IS  90 METERS

HEIGHT   WIND     DISTANCE CUMULATIVE    DROP      TERMINAL   TIME FOR CUMULATIVE
        DIR   SP  ADVECTED  DISTANCE     SIZE      VELOCITY   1 M FALL    TIME
  M          M/S        M          M          M        M/S          S           S

   1  329.2  .72     83.20      83.20 .1751E-04 -.861E-02     116.20    4136.33
  10  330.0 1.54    125.99    1152.25 .2090E-04 -.123E-01      81.58    3247.08
  20  330.9 1.95    119.34    2383.15 .2412E-04 -.163E-01      61.31    2532.00
  30  331.8 2.23    109.47    3522.08 .2695E-04 -.204E-01      49.12    1978.18
  40  332.6 2.45    100.55    4566.62 .2952E-04 -.244E-01      40.99    1526.00
  50  333.5 2.64     92.95    5529.27 .3189E-04 -.284E-01      35.17    1143.77
  60  334.4 2.81     86.53    6422.58 .3409E-04 -.325E-01      30.81     812.65
  70  335.2 2.96     81.06    7257.07 .3617E-04 -.365E-01      27.41     520.52
  80  336.0 3.09     76.36    8041.23 .3813E-04 -.405E-01      24.70     259.09
  90  336.9 3.22     72.27    8781.87 .4000E-04 -.445E-01      22.47      22.47
```

Table 9
Location C

```
THE HOUR IS 19

THE WIND IS  40 3

THE SPRAY HEIGHT IS  90 METERS

HEIGHT   WIND     DISTANCE CUMULATIVE    DROP     TERMINAL   TIME FOR CUMULATIVE
       DIR    SP  ADVECTED  DISTANCE     SIZE     VELOCITY   1 M FALL    TIME
  M          M/S         M         M         M         M/S          S          S

   1  39.19  .59     68.94     68.94 .1751E-04 -.861E-02     116.20    4136.33
  10  40.00 1.54    125.99   1088.55 .2090E-04 -.123E-01      81.58    3247.08
  20  40.89 2.06    126.29   2362.26 .2412E-04 -.163E-01      61.31    2532.00
  30  41.77 2.44    119.75   3590.78 .2695E-04 -.204E-01      49.12    1978.18
  40  42.65 2.75    112.60   4748.69 .2952E-04 -.244E-01      40.99    1526.00
  50  43.51 3.01    106.01   5837.81 .3189E-04 -.284E-01      35.17    1143.77
  60  44.36 3.25    100.16   6865.11 .3409E-04 -.325E-01      30.81     812.65
  70  45.21 3.47     95.02   7837.88 .3617E-04 -.365E-01      27.41     520.52
  80  46.04 3.66     90.49   8762.66 .3813E-04 -.405E-01      24.70     259.09
  90  46.87 3.85     86.47   9645.06 .4000E-04 -.445E-01      22.47      22.47
```

Table 10

Locations D,E

```
THE HOUR IS  6

THE WIND IS 240 7

THE SPRAY HEIGHT IS  60 METERS

HEIGHT   WIND      DISTANCE CUMULATIVE   DROP     TERMINAL   TIME FOR CUMULATIVE
        DIR   SP   ADVECTED  DISTANCE    SIZE     VELOCITY   1 M FALL    TIME
  M          M/S       M         M         M         M/S         S          S

  1   239.2 1.31     63.14      63.14  .2722E-04  -.208E-01     48.17    1929.06
 10   240.0 3.60    147.69    1162.82  .2952E-04  -.244E-01     40.99    1526.00
 20   240.9 4.89    171.83    2794.06  .3189E-04  -.284E-01     35.17    1143.77
 30   241.8 5.84    179.85    4564.07  .3409E-04  -.325E-01     30.81     812.65
 40   242.6 6.62    181.58    6375.43  .3617E-04  -.365E-01     27.41     520.52
 50   243.5 7.31    180.43    8186.55  .3813E-04  -.405E-01     24.70     259.09
 60   244.4 7.92    177.89    9977.67  .4000E-04  -.445E-01     22.47      22.47
```

References Cited

BERRY, E.X., and PRANGER, M.R.; (1974): Equations for Calculating the Terminal Velocities of Water Drops. J. Appl. Met. 13, 108-113.

DAVIS, C.N.; (1945): Definitive Equation for the Fluid Resistance of Spheres. Proc. Phys. Soc. London, A57, p. 259-270.

ELLIS, R.A.; (1981): Aerial ULV Application of Baygon MOS to Control Mosquitoes During the 1981 Outbreak of Western Equine Encephalitis in Manitoba. Internal Report, the City of Winnipeg Parks and Recreation Department, Insect Control Branch.

FRASER, H.M., and BELLAN, P.; (1970): Wind Profiles and Tower Shadow Measurements at the Nelson River Test Line, Manitoba. Atmospheric Environment Service, Environment Canada, (formerly Dept. of Transport, Meteorological Branch), TEC 743.

GOERING, C.E., BODE, L.E., GEBHARDT, M.R.; (1972): Mathematical Modelling of Spray Droplet Deceleration and Evaporation. Transactions ASAE 15, 220-225.

GOLDE, R.H. (ed); (1977): Lightning, Volume 2. Academic Press, New York.

HESS, S.L.; (1959): Introduction to Theoretical Meteorology. Holt, Rinchart, and Winston, New York.

HOLTON, J.R.; (1972): An Introduction to Dynamic Meteorology. Academic Press, New York.

KING, H.W., WISLER, C.D., and WOODBURN, J.G.; (1953): Hydraulics. 5th Edition. John Wiley and Sons Inc., New York.

MUKAMMAL, E.I., and MCKAY, G.A.; (1971): Synoptic Scale and Long-distance Transport of Pesticides. Meteorological Aspects of Pollution In Relation to Agricultural Pesticides, Bergsteinson, T.J., and Baier, W., eds., Research Branch, Agriculture Canada, Ottawa.

REID, J.D., and CRABBE, R.S.; (1980): Two Models of Long-range Drift of Forest Pesticide Aerial Spray. Atmospheric Environment, 14, P. 1017-1025.

STEEL, R.G.D., and TORRIE, J.H.; (1960): Principles and Procedures of Statistics. McGraw-Hill Book Co. Inc. New York.

TRAYFORD, R.S., and WELCH, L.W.; (1977): Aerial Spraying: A simulation of Factors Influencing the Distribution and Recovery of Liquid Drops., J. Agric. Eng. Res., 22, p. 183-196.

YOUMANS, T.; (1981): Mosquito Spray Program - Manitoba, August, 1981 - Baygon Droplet Monitoring. Internal Report, Environmental Protection Service, Environment Canada, Winnipeg.

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

Effectiveness of the Emergency Mosquito Vector Control Operations

R.A. Ellis, B.Sc., M.Sc., Ph.D. Entomology, R.P.E.

City of Winnipeg Parks & Recreation Department
Insect Control Branch, 2799 Roblin Boulevard
Winnipeg, Manitoba R3R 0B8

and

R.A. Brust, B.S.A., M.Sc., Ph.D. Entomology, F.E.S.C.

Department of Entomology
University of Manitoba
Winnipeg, Manitoba R3T 2N2

Abstract

Assessments made of the efficacy of the 1977 and 1981 emergency mosquito vector control operations in Manitoba, including the use of landing-biting rates, caged adult bioassays, and adult mosquito traps, are reviewed with particular reference to the Winnipeg control zone. The evidence suggests that there was a significant reduction in the *Culex tarsalis* Coq. population level following insecticide applications in both 1977 and 1981. Although the reductions were of short duration (i.e., 3-7 days), apparently they were sufficient to break the virus transmission cycle in those years. Because the data presented were collected on an "ad hoc" basis and, in some respects, could be considered inconclusive, several recommendations are made regarding ways in which to improve future evaluations of such emergency aerial ULV spray programs.

Numerous successful aerial ultra-low volume (ULV) insecticide applications, designed to reduce the threat of mosquito-borne disease in North, Central and South America, Africa and Asia, have been documented in the literature (e.g., Kilpatrick, 1967; Kilpatrick & Adams, 1967; Mitchell *et al*., 1969; 1970; Parker *et al*., 1972; Pant *et al*., 1974; Mathis *et al*., 1974; Brust & Ellis, 1976; Parker, 1978; Uribe *et al*., 1980; Carter *et al*., 1981; Pennington, 1981; Reeves, 1982). The degree of success is usually based on an entomological evaluation to measure reductions in vector mosquito activity and populations following treatment, epidemiological assessments rarely being made or reported.

The logic of such an approach is that, if there is a significant reduction (i.e., 80-95%) of mosquito vectors following aerial ULV applications, there will be a corresponding decrease in the risk of virus transmission.

Decreases in vector activity and population levels are usually measured using several complementary methods, including landing-biting rates, mosquito trap collections and caged mosquito bioassays (Anon., 1982). The overall reduction and duration is usually based on a review of the results of the various measures. Over the years, such assessments have developed in their sophistication to include concurrent studies of spray droplet size and dispersal, spray deposition, and the impact on non-target organisms and other components of the environment.

Unfortunately, many of the assessments made of emergency vector control programs have been incomplete or inadequate in some key area. Such deficiencies are often a result of inadequate contingency planning and funding, lack of expertise in some of the various disciplines involved and, of course, the emergency which often does not provide sufficient lead time for researchers to prepare, organize and carry out the necessary field studies. Considering the number of problems involved in an entomological assessment of an emergency vector control program, it is remarkable that so many good studies have been reported in the literature.

During the past 3 health emergencies due to Western Equine Encephalitis (WEE) in Manitoba (i.e., 1975, 1977 and 1981), entomological evaluations of the effectiveness of aerial ULV spraying with propoxur have been carried out. Although the 1975 evaluation (Brust & Ellis, 1976) demonstrated the value of such aerial applications, technological improvements in the spray equipment and insecticide formulation have been made (Ellis 1976, 1977, 1982b). This paper describes the assessments made in 1977 and 1981, concentrating on the latest emergency operations carried out over the City of Winnipeg.

Although this paper centers on an evaluation of the emergency aerial ULV application of insecticide for mosquito vector control at Winnipeg, Manitoba, in 1977 and 1982, it should be noted that 2 major vector control programs were carried out simultaneously in a coordinated and complementary fashion in both years. These programs are described elsewhere in detail (Ellis, 1977, 1982a, b). The insecticides and equipment used in these programs is outlined below (Table 1). The aerial spray operations are believed to have had more impact on the vector populations than the ground-based operations because the former provide more complete coverage of the treatment area.

METHODS AND RESULTS

Caged Adult Mosquito Bioassays:

Adult female mosquitoes of *Culex tarsalis*, *Culex restuans*, and *Culiseta inornata*, reared in the laboratory from field-collected larvae, were transferred from laboratory holding cages to the bioassay cages within 3 hours of being placed in the field for the tests. The bioassay cages were rectangular (25 x 150 mm) with 2 sides of 1.6 mm fibreglass screen and 2 sides and the ends of acrylic. One end was drilled to take a cork, enabling transfer of the mosquitoes.

In 1977, the bioassay cages were placed on 30 cm wooden stakes in open grassy areas, directly on the ground under grass cover and under shrubs to determine the mortality rates associated with degrees of vegetative protection. A mixture of the 3 above species was used in the bioassay cages which were located about 1 km apart, perpendicular to the flight lines of the aircraft. The entire Winnipeg spray block was treated in 1 day on August 14th, in 3 sections using the same number of spray flights. However, because the bioassay cages located in the central section suggested an unacceptable level of control, this particular section was subsequently retreated on August 17th (Table 2). Hindsight revealed that mid-day applications of insecticide may not provide an acceptable level of control. Only 66.2% mortality was observed in the central section, sprayed at mid-day, on August 14th, compared with 98.3% in one of the side sections, sprayed in the evening. Excluding the mid-day application, the average mortality in all 3 degrees of vegetative cover was 76.4% with the early morning flight and 74.9% with the evening flight.

Following the review of the 1977 bioassay tests, the methods were changed. One of the changes was to locate the bioassay cages in open, grassy fields to allow for a more realistic exposure of the caged mosquitoes to the air-borne spray droplets. The other change was to spray only when mosquitoes were active: i.e., the early morning and evening. Despite the cages being placed in more open areas, the mortality measured is probably still an underestimate of that occurring with the free-flying mosquitoes because the 2 screen sides and the 2 plastic sides undoubtedly reduce the chances of an insecticide droplet impinging on a mosquito inside the cage.

The 1981 bioassay results are summarized below (Table 3). Because of poor weather in early August, the 5 sections of the Winnipeg spray block were sprayed in a series of flights between August 4 and 11. Bioassays were carried out on 3 of the 5 sections. The percent mortality, 24 hours post-treatment, was 97.5 and 95.7% on August 4 and 7, respectively, and 71.6% on August 10th. Interestingly, the only instances of low mortality were on August 10 when some of the bioassay cages were either at the edge of a swath or under protective vegetative cover. Combining the percent mortalities of August 4th and 7th gives an average 96.6% control. Combining the mortalities of all 3 sections gives an average of 89.5%.

Landing-Biting Collections:

During the 1981 emergency spray operations, estimates were made of mosquito biting rates using the landing-biting collection method in one of the central sections of the Winnipeg spray block: i.e., mosquitoes coming-to-bite were aspirated from the front of a person dressed in blue denim over a 10 minute period, at 2 locations spaced 2 km apart, on several consecutive nights before and after spraying (Table 4).

The decline in the biting rate, from an average of 21.6 females/10 minute count before spraying to 0 immediately following spraying, was dramatic. In the 3 days following spraying, the counts averaged 2.1 females/10 minute count. On the fourth day, the average was back up to 20.5 females/10 minute count. Heavy rains, 2 weeks earlier, account for the return to high nuisance levels. The key point is that most of the infected and infective females present before spraying apparently were destroyed. The biting counts indicate that the virus transmission cycle was broken.

Mosquito Light Trap Collections:

In both 1977 and 1981, the City of Winnipeg's Insect Control Branch operated a network of 19 New Jersey style light traps, equipped with standard 25 watt bulbs, in (15) and around (4) Winnipeg. Daily collections, before and after treatment, were made. The data for August, 1977 and 1981, are tabulated below (Tables 5,6).

In 1977, the City was aerially sprayed completely in one day: i.e., August 14th. However, the central of 3 Sections, sprayed during the middle of the day showed poor results, according to caged mosquito bioassay monitoring, and was subsequently retreated on August 17th. Comparison of the mean numbers of mosquitoes inside and outside the aerial spray block indicates a sharp reduction in mosquito activity and levels following spraying over the City (Table 5). C. tarsalis females were not collected in the traps inside Winnipeg for 7 days.

Light trap data are useful in assessing spraying if there are comparative data from outside the spray block and if the spraying takes place over a 1-3 day period, as occurred in 1977. However, in 1981, because of repeated delays due to adverse weather, spraying took place between August 4th and 11th, a period of 8 days. Consequently, the mosquito trends are more difficult to follow (Table 6). Further, in 1981, as a result of extensive mosquito larviciding and adulticiding by the City of Winnipeg, prior to the declaration of a health emergency, mosquito levels within Winnipeg were 5 times less than those outside the City (e.g., August 1st-4th). Aerial spraying started on August 4th and the numbers of mosquitoes remained low for at least another 2 weeks inside the City. Outside the control zone, mosquito activity increased. One could conclude from this that aerial spraying further reduced the resident mosquito population in Winnipeg and kept it low until, on August 19th, a rapid resurgence of flood water Aedes spp. occurred. Following spraying, the low numbers of C. tarsalis collected in the traps were immediately reduced to zero and, thereafter, collected only occasionally and in very low numbers.

In 1981, New Jersey style mosquito light traps, equipped with 100 watt incandescent bulbs, were also used to monitor mosquito activity in 2 of the other municipalities included in the emergency spray operations: i.e., Virden and Portage la Prairie. The pre- and post-treatment mosquito levels are given below (Table 7). Both nuisance and vector species were reduced to low levels for 4-7 days following treatment. Heavy rainfalls, about 2 weeks before treatment, were responsible for the increased levels that subsequently occurred.

RECOMMENDATIONS TO IMPROVE FUTURE EFFICACY EVALUATIONS:

Several complementary components are needed in a thorough entomological assessment of the effectiveness of major WEE vector control programs. This matter was recently reviewed by an ad hoc subcommittee of the Manitoba Arbovirus Surveillance Committee (Brust *et al.*, 1982). The recommendations that were developed are presented below in an abbreviated form: i.e.,

1. Caged adult mosquito bioassays, using vector species, should continue to be used.

2. Mechanical mosquito traps, operated inside and outside the spray block, should also continue to be used.

3. Landing-biting collections of mosquitoes, before and after spraying, should also continue.

4. Funnel traps, mounted on vehicles, should be an added component, being operated on successive nights before and after the insecticide application.

5. Weather conditions and spray droplet size and dispersion should be assessed to assist in the interpretation of bioassay and collection data.

Inherent to all of these recommendations is an increased sample size, a greater number of replicates, and, adequate untreated controls for comparison purposes. These 5 recommended ingredients of a thorough assessment are considered essential. Time, funds and expertise permitting, each of the components could be expanded to provide additional important information; for example, landing-biting collections of female mosquitoes could be dissected to determine parous rates indicating "old" or newly-emerged females, a high percentage of "new" females after spraying indicating good control and vice versa.

Acknowledgements

The technical assistance of the following people in gathering and assembling the efficacy data is gratefully acknowledged: i.e., J.L. Buth, M.M. Chance, R.L. Cochrane, D. Delf, R. Dew, C. Moir and F.S. Richards.

References Cited

Anon., 1982. Emergency Vector Control after Natural Disaster. Pan American Hlth Org., WHO, Washington, D.C. 98 p.

Brust, R.A. and R.A. Ellis, 1976. Assessment of the emergency mosquito control operation in Manitoba, 1975. Can. J. Public Hlth 67 (Suppl. 1):69-71.

Brust, R.A. *et al.*, 1982. Report of the Manitoba Arbovirus Surveillance Committee's Ad Hoc Subcommittee on Effectiveness of Spraying for WEE control. Unpubl. Rpt., 14 p.

Carter, J.H. *et al.*, 1981. The operational response to the 1980 outbreak of Eastern equine encephalitis in southeastern Georgia. Mosquito News 41:785-789.

Ellis, R.A., 1976. Emergency measures and mosquito control operations during the 1975 Western encephalomyelitis outbreak in Manitoba. Can. J. Public Hlth. 67 (Suppl. 1):59-60.

Ellis, R.A., 1977. The 1977 outbreak of encephalitis in the Province of Manitoba. City of Winnipeg Unpubl. Rpt., 11 p.

Ellis, R.A., 1982a. Emergency mosquito vector control operations. (In press).

Ellis, R.A., 1982b. Emergency mosquito vector control by the City of Winnipeg. (In press).

Kilpatrick, J.W., 1967. Performance specifications for ultra-low volume aerial application of insecticides for mosquito control. Pest Control (May), 3 p.

Kilpatrick, J.W. and C.T. Adams, 1967. Emergency measures employed in the control of St. Louis encephalitis epidemics in Dallas and Corpus Christi, Texas, 1966. Proc. California Mosquito Control Assoc. 35:53.

Mathis, H.C. *et al.*, 1974. A helicopter application of ULV Dibrom against *Culex tritaeniorhynchus* in Seoul, Korea. WHO/VBC/74.501.

Mitchell, C.J. *et al.*, 1969. Effects of ultra-low volume applications of malathion in Hale County, Texas. 1. Western encephalitis virus activity in treated and untreated towns. J. Med. Ent. 6:155-162.

Mitchell, C.J. *et al.*, 1970. Effects of ultra-low volune applications of malathion in Hale County, Texas. II. Mosquito populations in treated and untreated towns. J. Med. Ent. 7:85-91.

Pant, C.P. *et al.*, 1974. Aerial spraying with malathion ULV using a single engine aircraft to control Aedes aegypti during an epidemic of Dengue haemorrhagic fever at Semarang, Indonesia. WHO/VBC/74.489.

Parker, J.D., 1978. Aerial spraying in public health. Agricultural Aviation 19(2):65-70.

Parker, J.D. *et al.*, 1972. Ultra-low volume applications of malathion for the control of *Aedes simpsoni* and *Aedes aegypti* in East Africa. WHO/VBC/72.411.

Pennington, N.E., 1981. Eastern equine encephalitis: Michigan, 1980. Proc. Ontario Mosquito Control Assoc., 10 p.

Reeves, W.C., 1982. A memorial to Findlay, Reed, Gorgas and Soper as major contributors to present day concepts essential for control fo mosquito-borne arboviruses. Mosquito News 42:313-319.

Uribe, L.J. *et al.*, 1980. Aerial ULV application trial of malathion against *Aedes aegypti* in the City of Colombia.WHO/VBC/80.768.

Table 1. Insecticides and application rates used in the 1977 and 1981 emergency mosquito vector control programs at Winnipeg.

Insecticide (Product Name)	Spray Equipment Used	Application Rate (a.i./ha)
Adulticiding Program:		
Propoxur (Baygon MOS)	DC-6B/ Beecomist	438.4 ml/ha (52.5 g a.i./ha)
Propoxur (Ultrafog ULV)	Leco HD Cold Fogger	190 ml/ha (22.8 g a.i./ha)
Malathion (Riddex Cythion)	Leco HD Cold Fogger	20 ml/ha (22.9 g a.i./ha)
Methoxychlor (Methoxychlor 2.4 EC)	TIFA Thermal Fogger	1.05 l/ha (50.4 g a.i./ha)
Malathion (Malathion 50EC)	Buffalo; Myers; Portables	1.12 l/ha (560 g a.i./ha)
Larviciding Program:		
Chlorpyrifos (Dursban 2.5G)	Bell 47G4A; Buffalo	2.24 kg/ha (56 g a.i./ha)
Chlorpyrifos (Dursban 2E)	Buffalo; Myers; Portables	70.2 ml/ha (26.9 g a.i./ha)
Mineral Oil (Flit MLO)	Buffalo	55.3 l/ha (45.66 kg a.i./ha)

Table 2. Bioassays of 3 separate aerial ULV applications of Baygon during 1977 and 2 sections of Winnipeg using a mixture of *C. tarsalis*, *C. restuans* and *C. inornata*.

		Percent Mortality[1]		
Date	Cage	Above Tall Grass	In Tall Grass	Under Shrubs
August 14 (early a.m.)	1	100	100	70
	2	100	100	80
	3	100	90	40
	4	100	100	0
	5	100	20	-
	6	90	90	-
C[2]=15/60		$\bar{X}$ = 98.3	$\bar{X}$ = 83.3	$\bar{X}$ = 47.5
August 14 (mid-day, central section)	1	30	20	0
	2	50	0	60
	3	60	20	20
	4	100	20	30
	5	100	0	70
	6	80	80	0
	7	60	-	30
	8	50	-	-
C=15/60		$\bar{X}$ = 66.2	$\bar{X}$ = 23.3	$\bar{X}$ = 30
August 17 (evening, central section retreated)	1	100	50	40
	2	90	90	0
	3	100	100	90
	4	100	60	-
	5	100	100	-
	6	100	100	-
C=15/60		$\bar{X}$ = 98.3	$\bar{X}$ = 83.3	$\bar{X}$ = 44.3
Overall Mean Mortality		87.6	63.2	40.3

[1] At 24 h post-exposure

[2] C=mortality in untreated, control mosquitoes.

Table 3. Bioassays of 3 separate aerial ULV applications of Baygon during 1981 over 3 sections of Winnipeg using caged adult females of Culex restuans (N=10/bioassay cage; 1 cage/site).

Site No.	Percent Mortality (%) [1] Aug. 4	 Aug. 7	 Aug. 10
1	100	100	50[2]
2	100	100	100
3	100	100	90
4	100	90	100
5	100	80	20[3]
6	100	100	70
7	90	100	
8	90		
Mean	97.5	95.7	71.6
Control	0 n = 60	0 n = 40	0 n = 50

Combined % mortality (Aug. 4, 7 and 10): 88.7

1/ At 24 hours post-exposure.
2/ Site near the edge of the spray swath.
3/ Site under a tree canopy. All the other sites were in open, grassy areas.

Table 4. Pre-spray and post-spray landing-biting counts of mosquitoes at 2 sites in Winnipeg, August, 1981.

	Collection Site		Weather Condition (2200 h)	
	A (2130 h)	B (2200 h)	Temp.°C	Rel. Hum. (%)
Pre-Spray				
Aug. 6	31[1]	28	16	79
7	16	19	18	83
8	20	23	13	78
9	19	17	15	78
Post-Spray				
10	0	0	19	79
11	0	2	20	79
12	1	1	17	79
14	4	5	15	77
15	22	19	14	66

1/ Based on 10 minute counts on a human host.

2/ Aerial spraying conducted at 1900 h on August 10 over collection site areas.

Table 5. Pre- and post-spray light trap collections of mosquitoes inside and outside the Winnipeg spray block during August, 1977.

August Date	Mean No. Female Mosquitoes/Trap/Night [1]	
	Inside Spray Block [2]	Outside Spray Block [3]
1	11.5	67.0
2	24.9	82.2
3	11.5	79.5
4	11.0	26.2
5	14.2	25.7
6	14.3	17.0
7	17.9	35.7
8	23.2	43.0
9	56.4	105.0
10	6.6	23.0
11	1.3	3.5
12	11.1	63.2
13	2.2	3.0
14[4]	5.4	19.5
15	7.0	30.7
16	1.2	13.2
17[4]	1.8	4.2
18	1.0	4.7
19	1.6	9.5
20	3.9	6.0
21	4.6	1.5
22	3.5	2.0
23	0.8	2.2
24	3.0	3.2
25	5.8	9.2
26	5.2	17.0
27	6.2	13.2
28	6.2	16.0
29	0.8	2.5
30	3.0	20.7
31	3.4	8.2

1/ Standard New Jersey style mosquito light traps, equipped with 25 watt incandescent bulbs, collected on mornings of dates shown.

2,3/ Based on 15 traps inside and 4 traps outside the Winnipeg spray block.

4/ All 3 sections of Winnipeg were sprayed on August 14th; the central section was retreated on August 17th based on the bioassays results for that section.

Table 6. Pre- and post-spray light trap collections of mosquitoes inside and outside the Winnipeg spray block during August, 1981.

August Date	Mean No. Female Mosquitoes/Trap/Night[1]	
	Inside Spray Block[2]	Outside Spray Block[3]
1	5.2	30.0
2	6.2	37.2
3	5.5	44.5
4	1.6	12.2
5	6.3	54.3
6	2.8	41.5
7	6.6	187.0
8	3.9	36.5
9	3.7	79.8
10	4.1	53.0
11[4]	1.3	20.3
12	2.1	38.0
13	2.6	10.2
14	3.0	32.0
15	2.0	7.3
16	2.1	4.5
17	3.4	10.0
18	8.8	22.0
19	40.9	55.5
21	119.1	93.3
22	54.2	89.7
23	28.1	152.5
24	13.8	52.3
25	31.3	105.0
26	15.4	94.5
27	14.7	24.8
28	8.4	23.7
29	10.4	47.0
30	16.5	110.8
31	22.6	72.3

1,2,3/ As per Table 5 footnotes.

4/ The 5 sections of Winnipeg were treated once between August 4th and 11th.

Table 7. Pre- and post-spray light trap collections of mosquitoes at 2 urban centres in Manitoba during August, 1981.

Urban Centre	August Periods	Total No. Mosquitoes	*C. tarsalis* No.	Mean Daily Temperature (°C)
VIRDEN	Pre-Spray			
	5-7	17	4	20-23
	8-10	22	6	17-22
	11-12	50	1	20-22
	13-14	9	2	18-22
	15[1]	1	0	16
	Post-Spray			
	16-18	5	2	17-21
	19-20	30	3	20-23
PORTAGE LA PRAIRIE	Pre-Spray			
	2-3	298	8	19-21
	4-5	225	40	19-21
	6-7	229	42	21-22
	8-10	369	34	17-20
	Post-Spray			
	11[2]-14	72	6	17-24
	15-17	72	1	15-22
	18-19	30	0	22
	20-21	140	1	22
	22-24	370	2	21-23
	25-27	465	2	20-22

1,2/ Virden & Portage were aerially sprayed on August 15th and 11th, respectively.

CHAPTER VIII:

Safety Evaluation of Insecticides

An Explanation of the Pre-registration Safety Evaluation of Insecticides Registered in Canada

J.E. Hollebone, M.S., Ph.D., D.I.C.,
Pesticides Division, Agriculture Canada, Ottawa, Ontario,
K1A 0C6

Abstract

Pesticides are regulated in Canada by Agriculture Canada under the Pest Control Products Act. The role of other departments in the regulatory review process and the data requirements before registration are discussed. Compliance and re-evaluation functions are briefly discussed.

The aerial use of propoxur over an urban centre has raised questions regarding the safety of propoxur to humans exposed during the Manitoba spray program. The toxicity of propoxur has been reviewed by toxicologists at Health and Welfare twice, first during the initial registration review and again within the last year for the NRC document on the "Effects of Propoxur on Environmental Quality (NRCC, 1982)".

One of the major concerns arising from the 1982 Manitoba workshop and again at the Manitoba Clean Environment Hearings seems to be that pesticides are allowed into the market with a minimal safety review or no review at all. Because the requirements for data are in fact very extensive and are estimated to cost the company seeking registration in the order of $10 - 15 million, the remainder of this paper will attempt to correct the misperception and detail the pre-registration requirements in some detail.

All pesticides must be registered under the Pest Control Products Act before being offered for sale in Canada. Although Agriculture Canada administers the Act, broad consultation is sought with other government departments such as Health and Welfare Canada, Environment Canada, and Fisheries and Oceans Canada before registration. Other experts in the provinces, universities and research scientists within the Department may also be consulted. The review process is not static but is constantly being updated and modified as technology changes and new knowledge becomes available.

Pre-registration requirements are set out in a series of trade memoranda (T-1-212, 223, 232) available to the public upon request from the Pesticides Division of Agriculture Canada. The registrant of a product containing a new active ingredient is required to submit "a full data package". This supporting data will include chemical specifications, toxicology data to indicate hazard to user, bystander, via food residues or exposure, environmental chemistry and toxicology to determine safety to the environment, and metabolism data to determine the fate in target organisms. A formulated product of that active ingredient must be further supported by chemical specifications and acute toxicology on the product, residue and efficacy data.

Chemical specifications will include data on chemical properties, mode of manufacture, identification of starting materials, final products, and impurities identified to 0.1 ppm, analytical methodology, and samples of the technical and formulated products.

Toxicological requirements are detailed in Tables 1 and 2 and are requested to delineate the toxicity of a pesticide and its potential hazard to humans. (Table 1,2). Both technical material and the formulated products must be tested. This is to gain some insight into whether the inert ingredients have an effect on the toxicity of the product. The assessment of hazard resulting from use or bystander exposure is determined from exposure levels, the mode of entry either measured or estimated of toxin into the body, the magnitude of the absorbed dose, and the toxic action of the pesticide. From the toxicity studies conducted on a variety of species of animals, the no-effect level (NOEL)can be determined, and from the exposure studies, the maximum exposure can be measured. The margin of safety (MOS) between these two levels is determined as

$$\text{MOS} = \frac{\text{NOEL}}{\text{EXPOSURE}}$$

The acceptability of a margin will depend upon the severity and reversibility of the toxic effect. Historically, a margin of 100-fold has been accepted for many toxic effects. This allows for a factor of 10 for extrapolation from animals to man, and a factor of 10 to allow for differences in sensitivity from one person to another. However, much larger factors (e.g., up to 5,000) have been used when the effects are more severe.

Environmental requirements include studies which will determine the fate of the product in both the terrestrial and aquatic environmental, and it toxicity to birds, mammals and nontarget organisms, such as bees. Hazards to the aquatic organisms are also carefully reviewed. (Table 3).

Efficacy requirements include laboratory and field data which show that the product works under conditions of use. Data are also required to indicate possible phytotoxic effects upon vegetation.

The product label details the acceptable conditions for use, the safety precautions, environmental limitations, and disposal instructions. If the label is followed carefully, the product is considered safe for the conditions of use.

The evaluation process does not end with registration. Agriculture Canada Field Inspection staff monitor products in the marketplace to ensure continued compliance. Complaints and instances of misuse are reported and investigated.

A second major post-registration function is the periodic re-evaluation of active ingredients. As available technologies change attempts are made to modernize evaluation standards. A natural consequence of evaluation is that previously registered pesticides constantly fall behind the more modern registration standards. Pesticides registered as recently as 4 to 5 years ago with supporting data that was considered fully satisfactory at that time do not meet current standards. A large number of the older biting fly products fall into this category (e.g., products containing such active ingredients as methoxychlor, malathion and pyrethrin). Agriculture Canada is attempting to handle this problem in two ways: 1) through re-evalution of certain groups of older pesticides; and 2) by identifying information gaps in the technical data base supporting the registered product and requiring updated chemical specifications on each active ingredient as an important first step in bringing the older registered pesticides up to modern standards.

A re-evaluation may occur on a group of products which have generally been in use for some years, and some users, few or many, may have become dependent upon them in their particular operation. The activities are frequently no longer under patent and are therefore not protected. There is usually more than one basic manufacturer. Under these circumstances, it is generally not economical for the industry to produce quantities of expensive data; i.e., they will not see a return on their investment. The problem then becomes one of balancing benefits against data gaps as much as against identified risks, or alternately to find other sources to fund the expensive research that must be carried out to fill the gaps.

A final important role in pesticide regulation is the continuing development of updated guidelines for registration requirements. Toxicology guidelines have recently been published with a request for comments before finalization, (Registration Memorandum R-1-211). Others are being developed and include requirements for biological control agents, livestock pesticides, environmental quality and environmental fate, non-target organisms, and disposal and decontamination.

The role of Agriculture Canada in pesticide registration can be summed up by saying that infallible assurances of safety regarding any pesticide cannot be provided to the public. Agriculture Canada as administrators of the Pest Control Products Act is appreciative of the public concerns regarding pesticides. In administering regulatory responsibilities, the Department tries to assure safety, merit and value of newly registered products to "state of the art" technology. Attempts are being made to bring older products up to these standards. Regulators must continue to be guided by the scientific evidence to support regulatory decisions and by expertise from all levels as provided in the present system.

References Cited

NRCC (National Research Council of Canada). 1982. Effects of propoxur on environmental quality with particular reference to its use for control of biting flies. Associate Committee on Scientific Criteria for Environmental Quality, National Research Council of Canada, Ottawa, Canada. NRCC No. 18572, 237 pp

Table 1

GUIDELINES FOR PESTICIDE TOXICOLOGY DATA

	TECHNICAL	FORMULATION	SUGGESTED SPECIES	SEX	COMMENTS
Acute (including NOEL where applicable)	Oral LD_{50}	Oral LD_{50}	Rat or Dog	M & F	
	Dermal LD_{50}	Dermal LD_{50}	Rabbit or Rat	M & F	
	Inhalation LC_{50}	Inhalation LC_{50}	Rat	M & F	
	Primary Dermal Irritation	Primary Dermal Irritation	Rabbit	M or F	If required
	Dermal Sensitization	Dermal Sensitization	Guinea Pig or Rabbit	M	
	Delayed Neurotoxicity		Chicken	M	For ChE inhibitors
	Antidotes				
Short term	90-day Oral with Recovery Studies		Rat and Dog	M & F	
	12-month Oral		Dog	M & F	May be combined with 90-day study
	90-day Dermal with Recovery Studies	21-day Dermal	Rabbit or Dog	M & F	
	90-day Inhalation with Recovery Studies	21-day Inhalation	Rat	M & F	
	Delayed Neurotoxicity if Positive in Acute Tests		Chicken or Mammal	F M & F	

Table 2

GUIDELINES FOR PESTICIDE TOXICOLOGY DATA

	TECHNICAL	SUGGESTED SPECIES	SEX	COMMENTS
Long Term	Chronic Feeding	Rat	M & F)	Studies may be combined
	Oncogenicity	Rat	M & F)	
	Oncogenicity	Mouse	M & F	
Special Studies	Pharmacokinetic Studies - absorption, distribution, excretion and metabolism	Appropriate to Route Being Studied		Details to be discussed
	In vitro Mutagenicity			
	a) Point Mutation - microbial - mammalian			Positive results may indicate in vivo studies
	b) Chromosomal Abberations			
	c) DNA Repair			
	Teratogenicity	2 of: Rat Mouse Hamster Rabbit		
	Multi-generation Reproduction	One of species used for teratogenicity study, preferably Rat		2 generation, 4 litter study as minimum
		Exposure Studies		Done at the time of efficacy trials

* Sub-acute and chronic toxicity studies on environmental breakdown products may be required depending on quantities and significance (e.g. significant plant metabolites which do not occur in animal metabolism studies).

Table 3

GUIDELINES FOR ENVIRONMENTAL DATA REQUIREMENTS

I Environmental Chemistry

Physicochemical degradation (hydrolysis, photodegradation)
Metabolism (soil, aquatic, microbial)
Mobility (leaching, volatility, adsorption, desorption, water dispersal)
Field dissipation (foliage, litter, soil, water) (include analytical methods)
Accumulation
Disposal, storage, decontamination

II Environmental Toxicology

1. Birds and Mammals (Wild)

Acute Toxicity on 2 species
Short Term
Special Studies— long term seudies, reproduction, simulated or actual field testing, special metabolism studies etc.

2. Aquatic Organisms

Acute Toxicity on 2 Fish, and several invertebrate species
Short Term
Special Studies - long term studies, reproduction studies, simulated or actual field testing, special metabolism studies, etc.

3. Non-Target Invertebrates

Predators
Parasites
Bees
Earthworms
Soil microorganisms
Other soil, fresh water and marine invertebrates as applicable.

Monitoring of Pesticide Effects on Humans by Cholinesterase Testing

R. Hoare Ph.D.

Manitoba Health Services Commission
Cadham Provincial Laboratory 750 William Avenue
Winnipeg, Manitoba R3C 3Y1

Abstract

A serum cholinesterase level can be used as a measure of cholinesterase inhibition due to exposure to high levels of pesticides, or due to a congenital deficiency of the enzyme. In healthy individuals the effects are reversible, but in individuals with atypical cholinesterase levels, the risk of neurological complications is increased. Programs for the monitoring of cholinesterase levels in humans exposed to pesticides have been set up for the City of Winnipeg since 1976 and for licensed applicators in the province since 1981. All positive tests (levels decreased greater than 50% from individual's baseline values) are referred through private physicians. Results would indicated that a monitoring program is valuable in providing a safety factor for any individual at risk in the use of pesticides.

Serum cholinesterase deficiency may be congenital or acquired. Those individuals with a congenital deficiency have an abnormal cholinesterase, of which several genetic variants have been reported (Garry, P.J. 1971).

Serum cholinesterase assay is the best screening test for cholinesterase deficiency. If abnormally low values are found, it is then necessary to perform inhibition assays using dibucaine and fluoride to distinguish congenital deficiency from acquired deficiency of the normal enzyme. Acute or chronic liver disease is the most frequent etiology for acquired deficiency.

Cholinesterase is also decreased in organophosphate poisoning (Young, D.S. 1975). This affects both red cell and plasma enzyme levels. Some organophosphates are inhibitors of cholinesterase. This allows the overproductions of acetylcholine at nerve-muscle junctions. Symptoms will include muscle twitching, cramps and weakness; parasympathetic effects such as pinpoint pupils, nausea, sweating, diarrhea, and salivation; and various central nervous system effects. Laboratory diagnosis is based on finding decreased cholinesterase levels in erythrocytes or plasma. Although erythrocyte levels reflect poisoning more accurately than do plasma levels, which can be affected by many conditions and drugs, screening tests are generally based on plasma, measurement and levels within physiological ranges for a specific individual (baseline values) generally rule out anticholinesterase toxicity.

With reference to specific chemicals used for spraying such as propoxur (Baygon), evidence (Anon, 1982), has been shown that the anticholinesterase effects are reversible and in healthy individuals, no permanent effects were observed, however, no data was presented on the effects of the anticholinesterase agents on those individuals with atypical cholinesterase levels, or genetic deficiencies. These individuals would be considered at risk when exposed to organosphosphorus compounds.

HISTORICAL BACKGROUND

The monitoring of cholinesterase levels of individuals exposed to insecticide spraying was first initiated in Manitoba at the Provincial Laboratory unit, then located in Brandon during 1971 and 1973 to monitor the City of Brandon's spray personnel. This program concerned the spray teams operated by the City of Brandon. This laboratory was discontinued in 1974 due to the closing of Brandon's laboratory under Provincial operation. However, the monitoring program was reactivated in Winnipeg at the Cadham Provincial Laboratory, at the request of the City of Winnipeg, Insect Control Branch, and a regular monitoring scheme has been set up to continue on an annual seasonal basis. Baseline levels of cholinesterase are established each year, and levels are monitored bi-weekly throughout the spraying period.

A request from Manitoba Agriculture for the monitoring of cholinesterase levels of Provincial and private applicators of pesticides has resulted in the start of a province-wide program for all individuals directly connected with the use of insecticides in rural areas. This program was started in 1980 with minimal response from individuals and groups and is slowly gaining momentum as an ongoing program throughout all the areas involved.

MATERIALS AND METHOD

The cholinesterase method now being used at Cadham Provincial Laboratory, is an adaption of the Ellman procedure (Dietz, A.A. 1973). In our laboratory it has been adapted to the use of the centrifugal analyzer (Multistat III).

The Dietz method reports cholinesterase values as (I Unit = mol/ml serum/hour) with a normal range 4.9 to 12.0 I.U.

RESULTS AND DISCUSSION

The Provincial applicator testing program was accepted better in 1982 due to efforts to acquaint those involved with the importance of regularly scheduled monitoring periods over the time of spraying. However, the numbers tested as percentage of total (Table 1) and the compliance shown (Table 1), as an average of 58% leaves a lot of room for some improvement in the program. No positive results directly due to spraying were recorded although one individual with a low baseline level was found to be atypical, and therefore, highly at risk in working with pesticide materials. The individual was notified through his own physician, and adjustments were made in his contacts with pesticide to keep the risk at a minimum.

The results of the City of Winnipeg's Insect Control Branch, between 1976 and 1981 are shown below (Table 2). The mean positive findings are 0.5% of total tests, and these individual's levels, in all cases have returned to baseline or higher levels within a two week testing period. One individual who was not on the team in 1982, but showed a positive result in 1981 and 1979, was under medication for arthritis and hypertension, and his decreased levels could not be attributed directly to pesticide contact alone.

For a satisfactory screening program for individuals in contact with insecticides, a baseline value is obtained before exposure and blood samples are then taken at two week intervals during the period of insecticide use. Any levels below physiological range are repeated and a phenotype is determined. Individuals with atypical cholinesterase are considered at risk and referred to a physician for counselling concerning continuation of their contact with the insecticide. Normal individuals are cautioned and their levels are monitored. Removal from any contact with the organophosphate is necessary for a limited time in case of extreme inhibition.

It would appear that the inhibiting effect of pesticides on cholinesterase is generally reversible in normal healthy individuals providing the exposure is not cumulative and the individual at risk is removed for a period of one to two weeks from further spray contact. However, atypical heterozygotes with abnormally low levels could be at much higher risk than normal and a well run monitoring program should be in place for this type of individual, as well as for any group using pesticides. The short term effects of pesticides on cholinesterase are known, but the long term and cumulative effects will require many more years of data collection before definitive statements can be made. Perhaps the definition of a positive effect (i.e., 50% or greater decrease in cholinesterase level from baseline values) should be re-evaluated in assessing risk.

References Cited

Aldrich, F.D. 1969. Cholinesterase assays: Their usefulness in the diagnosis of anticholinesterase intoxication. Clin. Toxicol. 2:445.

Anon. 1982. Effects of propoxur on environmental quality. National Research Council Canada. NRCC 18572. 238 p.

Garry, P.J. 1971. Serum cholinesterase variants. Examination of several differential inhibitors, salts and buffers used to measure enzyme activity. Clin. Chem. 17:183.

Wetstone, H.J., Bowers, G N. Jr. 1963. Serum cholinesterase. Standard Methods of Clinical Chemistry. Publ. By Academic Press Inc. Vol. 4, page 57.

Young, D.S. et al 1975. Effects of drugs on clinical laboratory tests Clin Chem. 21(5) I.D.

Table 1

Areas of Licensed Applicator Cholinesterase Testing Program 1982

Area	Expected No. per area	Total test periods available	Actual test periods	Actual No. tested	Total tests	Compliance percentage
Altona	5	9	2	4	8	22
Beausejour	5	9	0	0	0	0
Grandview	3	9	6	3	5	67
Morris	6	9	8	5	32	89
Portage	6	9	7	6	27	78
Russell	3	9	7	3	16	78
Stonewall	3	9	2	3	6	22
Swan Lake	1	9	8	1	8	89
Swan River	3	9	6	3	16	67
Treherne	4	9	2	2	2	22
Winkler	8	9	6	4	17	67
Winnipeg	31	9	8	24	85	89
TOTALS	78	108	62	58	222	

Table 2

City of Winnipeg's Insect Control Branch
Cholinesterase Testing Program
1976 - 1981 (inclusive)

Year	N tested/year	Test periods	Total tests	Positive*	% Pos. Year
1976	42	9	378	1	0.3
1977	36	9	324	2	0.6
1978	38	9	342	1	0.3
1979	53	8	424	2	0.5
1980	28	9	252	0	0.0
**1981	39	10	390	1	0.3
TOTALS	236	54	2110	7	

* A positive change - 50% drop in cholinesterase level from baseline
** Change to Dietz method

Health Considerations in the Use of Insecticides in the Prevention of Western Equine Encephalitis

Peter Warner MD PhD FAPHA

Department of Health
Environmental Health Services
831 Portage Avenue
Winnipeg, Manitoba
R3G 0N6

Abstract

This paper examines the health considerations of the use of propoxur as an aerial spray in Manitoba for the prevention of Western Equine Encephalitis (WEE) and consists mainly of a review of information from a publication on propoxur published by the National Research Council of Canada.

It was concluded that, while pesticides must be regarded as dangerous toxic substances, there appears to be justification for the aerial spraying of propoxur in Manitoba in face of an epidemic of WEE.

Since, without extensive epidemiological evidence, it is not possible to determine the effect of aerial spraying on the spread of Western Equine Encephalitis (WEE), the evidence that it is or was effective must be indirect. In fact, the evidence rests solely on whether the aerial spraying significantly reduced the number of mosquitoes, particularly Culex tarsalis. If it can be shown that, in fact, Culex tarsalis were significantly reduced it would be reasonable to conclude that a significant proportion of Culex tarsalis infected with WEE virus were also reduced and thereby the number of human (and equine) cases of WEE were reduced.

If the conclusion is reached that Culex tarsalis is not significantly reduced by aerial spraying, than aerial spraying, under these circumstances is not justified.

If the conclusion is reached that Culex tarsalis is significantly reduced by aerial spraying, then the question becomes one of whether the harmful effects of the pesticide exceed those of an epidemic of WEE without the intervention of aerial spraying. WEE is a disease with high mortality and morbidity rates, and the question to be answered is whether aerial spraying of the pesticide has morbidity and mortality rates significantly less than those for the disease (it is to be noted that aerial spraying is not expected to affect the rates for WEE - theoretically, at least, if 50% of the mosquitoes were killed, the total number of cases of WEE would be halved but the same proportion of deaths and disability would be expected - in other words the rates of morbidity and mortality for WEE would remain the same).

The pesticide in question (propoxur or Baygon) was assigned by a joint committee of the World Health Organization and the Food and Agriculture Organization a Maximum "Acceptable Daily Intake" (ADI) of 0.02 mg ai/kg-bw based on a "no-effect level" of 0.2 mg ai/kg-bw - this is mentioned for future comparisons.

Most of the following information is derived from the publication entitled "Effects of Propoxur on Environmental Quality with particular reference to its use for Controls of Biting Flies" published by the National Research Council of Canada (NRCC, 1982).

This publication states that a crude estimate of a heavy exposure in a single application of 125 g ai/ha is 0.1 mg ai/kg-bw, which is one-half the no-effect level mentioned above (in Manitoba, the application was less than one-half of the above ie: 55 g ai/ha).

Propoxur is a reversible inhibitor of brain, central nervour system, erythrocyte and blood serum cholinesterases. Inhibition of cholinesterase reduces transmission of impulses in certain nerves and that is why it is called a nerve-poison but the effects of the poison are reversible.

In addition to anti-cholinesterase activity, the symptoms and signs of intoxication include pressure in the head, blurred vision, nausea, sweating, increased pulse rate and blood pressure, dizziness, abdominal pain, vomiting, diarrhoea, numbness of the face, pin-point pupils, pulmonary oedema and coma.

A 42-year-old male volunteer received a single oral dose of propoxur of 1.5 mg ai/kg-bw and in 15 minutes began to be affected by headache, blurred vision, nausea, sweating, but he started to feel better in another 30 minutes and ten minutes later was back to normal. The dose was 75 times the Acceptable Daily Intake.

Results of several studies in which humans were exposed to propoxur at 0.36 - 1.5 ai mg/kg-bw in single doses or 0.15 - 0.20 ai mg/kg-bw in 5 doses (total dose 0.75 - 1.00 ai mg/kg-bw) at 30 minute intervals (from 18 to 75 times the ADI) showed that:

i) Symptoms of carbamate poisoning could result from a single oral dose of propoxur from 0.2 to 0.4 mg/kg-bw.

ii) The reduction of *erythrocyte* cholinesterase to about 60% of the normal level is possible at doses lower than those in which signs and symptoms of carbamate poisoning are apparent. Recovery is extremely rapid (within two hours).

iii) *plasma* cholinesterase depression is insignificant at dosages as high as 1.5 mg/kg-bw hence depression of the activity of this enzyme would serve as a poor indicator of propoxur exposure.

A conclusion was that the detection of urinary phenolics (isopropoxyphenol) was a more satisfactory test of exposure to propoxur than tests for anti-cholinesterase. This information would be of imporcance for physicians.

In another trial exposure of four persons of propoxur to a domestic 2% formulation at a concentration of 3.0 ± 1.8 mg ai/m^3 for a period of 4 hours showed neither cholinesterase depression nor impairment of general health. Analysis of urine did reveal detectable amounts of the propoxur metabolites. The level of 3.0 mg ai/m^3 is six times greater than the Time Weighted Average and one and a half times greater than the Short Term Exposure Limit set by the American Conference of Governmental Hygienists.

Spray operators were studied after first, second and fifth weeks (with a 50% water dispersible formulation). The whole blood cholinesterase was depressed by about 10, 35 and 40% respectively. These returned to normal within 36 hours. It was not mentioned that any signs or symptoms resulted. There did not seem to be any cumulative effects.

A thirty-nine year old woman tried to commit suicide by drinking propoxur. She arrived in hospital in a coma, with pin-point pupils and frothing at the mouth. Her cholinesterase levels were normal (both blood cell and plasma) but propoxur and 2-isopropoxyphenol were found in the gastric aspirate and later in the urine. The woman recovered and is apparently well to the day of publication.

Eleven episodes have been described where operators (88) and general populations (34,477) have been involved. These occurred in Iran, Nigeria and El Salvador where propoxur was used to spray homes in concentrations of 1.5 to 2.0 g/m^2, more than 50 times higher than the application rates used for mosquito control in Canada. Signs and symptoms of propoxur poisoning included giddiness, nausea, headache, blurred vision, weakness, sweating, vomiting, pin-point pupils, dizziness, diarrhoea, abdominal pain, skin irritation, numbness or tingling of mouth, nose or face. Generally they disappeared in 24 hours and often within 3 hours. The proportion of people living in sprayed homes who complained varied from zero to 5% (average 0.5%). Of the 88 spray operators, 71 (81%) were affected with symptoms ranging from a mild burning sensation to those described above. No fatalities occurred and apparently there were no residual effects. In most cases overexposure of persons other than spray operators could be attributed to entering houses during or immediately after spraying, sweeping sprayed floors without wetting first and direct contact with sprayed surfaces. It was suggested that with a more cautious attitude these situations could be avoided. Also, the studies indicated that overexposure to propoxur of spray operators could be avoided by good personal hygiene, proper training, supervision, respirators and protective clothing.

The "no-effect" and ADI levels, the calculated exposure from spray, the work on human volunteers, the observations on field workers and population, all seem to indicate that the acute short term effects of propoxur were reversible and even with large doses no mortality was reported. Such adverse episodes as were described were either intentionally caused or appeared to be the result of badly conducted programs.

Because the rate of application in Manitoba was less than one-half of that quoted in the NRCC publication and 100 times lower than the programs in Iran, Nigeria and El Salvador, it would seem that the exposure of people to the aerial spray program in Manitoba is likely to have resulted in a negligible effect on them.

No chronic effects appear to have been described. In one episode skin rashes were reported in 5% to 200 people but subsequent investigation indicated that this was not a regular phenomenon.

As far as long-term effects are concerned, long-time dietary exposure studies with rats and mice do not indicate that propoxur is carcinogenic or mutagenic in animals.

It has been claimed that it is possible for propoxur to be "nitrosated" (particularly in the stomach) to form a substance of the nitrosamine class. Some nitrosamines are known to be carcinogenic but there are many nitrosamines some in foods and some can be detected in normal gastric juice.

While it can be said that a substance is carcinogenic, it is not possible to state, even with the best designed animal experiments, that a chemical, particularly a complex organic chemical, is not carcinogenic. However, it is claimed that if nitrosopoxur is formed at all, it is formed in small quantities and no evidence exists that it has caused cancer in humans.

To look at this in a slightly different way, WEE is a disease with at least 10% mortality and lifelong disability whereas, in 1980, deaths from malignant neoplasms from all causes in the USA were 0.1752%. It would not be unreasonable to conclude that the risk of death or lifelong disability is at least 100 times greater in WEE than with exposure to a speculative carcinogen.

Thus, if it can be asserted that aerial spraying with propoxur diminishes Culex tarsalis in significant numbers it is reasonable to conclude that WEE is diminished proportionately (such assumptions are not uncommon in medicine and in all types of research) then it is difficult to deny that aerial spraying is justified in the face of an epidemic of WEE.

It has been suggested or even directly stated that there is a vast lack of knowledge about pesticides to the extent that it could be considered irresponsible to recommend their use. In addition, others have issued very serious warnings about the toxicity of pesticides. It cannot be denied that there are gaps in knowledge nor that pesticides in general are toxic nor, if one catalogues all the toxic effects and all the adverse episodes relating to pesticides used or misused, that they are very dangerous and frightening chemicals. But, in the specific case of propoxur, which has been in existence for nearly twenty years, not only does there appear to be a considerable amount of information but also there appears to be justification that, if used as directed, in an occasional aerial spraying program, the risk of toxicity is very small.

Finally, it has been suggested that because the mortality of WEE is only 2% of all those infected (not all those infected actually contract the disease, case-mortality is the proportion of those who contract the disease and die and would be much higher than two per cent and is stated to be 10%) and that, by allowing those who become infected to become naturally immune to the disease, the population would build up its resistance to WEE with the necessity of an aerial spraying prognam. However, this suggestion was accompanied by a requirement that the annual mosquito abatement program would be continued. Quite apart from the possibility that only 20% of the population would be immunized, the annual use of pesticides raises questions in addition to those that are dealt with in this paper.

In conclusion, while chemical pesticides must be regarded as dangerous toxic substances, there appears to be justification for the aerial spraying of propoxur in Manitoba in face of an epidemic of Western Equine Encephalitis.

References Cited

Anon. 1982. Effects of Propoxur on Environmental Quality with Particular Reference to its Use for Control of Biting Flies. National Research Council of Canada, NRCC 18572, 238 p.

The Effects of the 1981 Manitoba Emergency Mosquito Control Program on Honey Bees[1]

D. P. Dixon, B.Sc. (Hons.), M.Sc.
B. G. Fingler, B.S.A., M.Sc.

Manitoba Agriculture
911 Norquay Building
Winnipeg, Manitoba
R3C 0V8

Abstract

Observations of honey bee colonies, experiments with caged bees and reports from beekeepers indicated that honey bees were killed by propoxur applications during the 1981 Manitoba Emergency Mosquito Control Program. Increased aggressiveness by bees and the disruption of normal honey bee behaviour were also observed following the spray applications. Beekeepers reported losses totalling $87,455.00 resulting from the effect of the spray on their colonies.

During the summer of 1981 the Manitoba beekeeping industry was comprised of approximately 1,550 beekeepers operating a total of 103,000 honey bee colonies. The total value of the honey and beeswax produced in 1981 was estimated at approximately $10.7 million, at the producer level. An aerial application of the insecticide propoxur was carried out over 21 communities in southern Manitoba. Because most of the locations where the insecticide was applied included agricultural as well as urban areas, there were large numbers of honey bee colonies found within many of the spray areas.

MATERIALS AND METHODS

During the emergency spraying, Manitoba Agriculture staff attempted to monitor the effects of the propoxur applications on honey bees by making observations at apiaries within the spray areas, conducting caged bee experiments and by receiving reports from beekeepers. Weight change data from honey bee colonies on scales within some spray areas was received from 3 beekeepers and from the University of Manitoba. Caged bee trials were conducted using mosquito cages with fibreglass window screening on two sides and with cylindrical cages made of coarse hardware cloth (3 mesh/cm). At each location 10 or 20 bees were placed in each cage and several cages were placed in the spray area and at least one cage with bees was placed outside of the spray area as a control. Cages were attached to a stake so that they were suspended about 1 m above the ground. All cages were placed in open areas with no canopy. Immediately following the spraying at Virden, bees were collected from sunflowers and caged for later observation. Mortality of these bees after 3 hours was recorded.

---

[1] This paper is a resume of a Manitoba Agriculture Report, with the same title, prepared in October, 1981.

At Morris a comparison of honey production in screened versus unscreened colonies was performed. Four days before Morris was sprayed, the honey was removed from all colonies and empty honey supers were put on the colonies. On the day of the spraying several colonies had their entrances screened all day to prevent bee flight and most of the colonies were left open. The apiary was sprayed at approximately 2000 hrs. The screens were removed at 2200 hrs and all colonies in the yard were left undisturbed until 1 September, 1981 at which time all of the honey supers were removed and the weight of extracted honey from 3 experimental (screened) and 5 control (unscreened) colonies was determined.

To encourage beekeepers to supply information on their observations of the effect of the spray on their colonies, a questionnaire was sent to all registered beekeepers in Manitoba.

RESULTS

Observations at Apiaries: The highest levels of honey bee mortality were observed at locations where the spraying took place in the late afternoon or early evening. Large numbers of dead bees were also observed at locations that were sprayed in the morning when the bees were flying. We observed up to 500 g (i.e. 4,000 - 6,000 bees) of dead bees at hive entrances in Winnipeg, Morris, Steinbach, Morden, Winkler, Virden and Beausejour. We also received reports from beekeepers of large numbers of dead bees at Selkirk, Altona, Carman and Swan River.

Besides killing or disabling the bees, we also observed that the spray illicited extremely aggressive behaviour by the bees immediately following the spray. When this happened people and animals close to a sprayed apiary were stung by bees at a much greater frequency than would occur normally. We observed this type of behaviour at Winnipeg, Morris, Virden, Beausejour and Dauphin.

Scale Colony Records: Scale colony data supplied to us indicated a weight loss of approximately 2 - 6 kg on the day of, or the day following spraying. Scale colony data from the University of Manitoba was inconclusive since a weight loss trend had begun before the spraying took place. Scale colony data from Dauphin is interesting since a net reduction in weight of 4 - 6 kg was observed on the day of spray even though very small numbers of dead bees were observed. The scale colony information also indicated a recovery in normal weight gain 1 - 2 days following the spray.

Results of Caged Bee Trials: Table 1.

TABLE 1. EFFECTS OF AERIAL APPLICATIONS OF PROPOXUR ON CAGED HONEY BEES DURING AUGUST, 1982

Location	Time Of Observation (h)[1]	No. of Cages		% Mortality		Range In % Experimental Mortality Between Cages
		Experimental	Control	Experimental	Control	
Floodway to Lagimodiere (Winnipeg)	12	6	3	16.7	0	0-80
Highway 90 Strip (Winnipeg)	12	9	5	16.9	.83	0-80
Stonewall	12	9	1	8.33	0	0-30
Gimli	3	5	2	52	0	0-100
Beausejour	12	12	1	5.8	0	0-30
Brandon	3	9	3	0	0	0

[1] Number of hours after the spray when mortality was recorded.

Observations at Virden: Bees that were collected and caged immediately following the spray were observed after 3 hours. The mortality observed was 77.8% in cage one and 85.7% in cage two. Approximately 4 hours after the area was sprayed, we visited the sunflower field and looked for dead bees but could find none that had died recently. We also visited an apiary about 1.5 km from the sunflower field and found several hundred dead and dying bees at each hive entrance.

Comparison of Unscreened Sprayed Colonies with Screened Sprayed Colonies: Following the removal and extraction of honey from the experimental and control colonies, it was determined that the average honey production of the unscreened colonies was approximately 47 kg and the average production of the screened colonies was approximately 41 kg. An unpaired t-Test indicated that there was no significant difference between the treatments in the mean amount of honey produced.

Observations of Beekeepers: As a result of returned questionnaires, 67 beekeepers indicated losses from the spray in 15 of the spray areas. The total number of colonies that were reported damaged by the spray was 3,725 and the estimated value of damage resulting from lost honey production, moving of colonies and extra winter feed required, amounted to approximately $87,455.00. Most of these beekeepers reported losses of about 1 super of honey (15 - 25 kg) per colony of bees in the spray area.

DISCUSSION

From our observations and those of beekeepers, it was concluded that the method of application of propoxur used during the 1981 emergency abatement program was responsible for killing large numbers of honey bees when the spray was carried out while bees were flying. Observed sublethal effects of the spray included: (1) increased aggressiveness by bees and (2) the disruption of normal colony behaviour for at least 1 day following the spray application.

The large range in mortality observed in the caged bee experiments may have been the result of variations in insecticide dispersal within the spray areas. Any uneven distribution of the insecticide may put free flying bees at a higher risk as they could fly into areas of high insecticide concentration.

The observations made at Virden indicated that the insecticide did not kill bees immediately and that many were probably able to return to their hives, or the vicinity of their hives before dying.

Beekeepers in some areas probably lost significant amounts of honey production as a result of the spray. Because of the lack of accurate forewarning of when each area would be sprayed it was very difficult for beekeepers to attempt on-site protection of their bees.

The results of experiments at Morden, which indicated no significant

difference in honey production between screened and unscreened colonies, may have resulted from one or more of the following: i.e.,
(1) there was a significant loss of honey production in the protected colonies during the period of confinement;
(2) the insecticide affected the screened colonies the day following the spray after the screens had been removed;
(3) the screened colonies suffered significant stress during confinement that later reduced honey production; and/or
(4) the insecticide had little overall effect on honey production.

More research is required to better understand the economic impact of this type of insecticide application on beekeepers.

ACKNOWLEDGEMENTS

We wish to express appreciation to the many Manitoba beekeepers who supplied information and provided access to their beekeeping operations, to D. Hooge for technical assistance and to Dr. S. C. Jay for reviewing the manuscript.

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

Analysis of Water Supplies Following the Aerial Application of Baygon

K. W. Plews, B.S.A. MSc.
Agrologist, Manitoba Environmental Management Division,
Environmental Control Services,
Box 7, Building 2 - 139 Tuxedo Avenue,
Winnipeg, Manitoba.
R3N OH6

T. Youmans, BSc.
Environmental Officer, Environment Canada,
Environmental Protection Service,
800 - 275 Portage Avenue,
Winnipeg, Manitoba.
R3B 2B3

Abstract

Water analysis were carried out at 21 locations in Manitoba following the aerial application of Baygon in August, 1981. The results indicated that Baygon levels were insignificant and short-term, posing no significant adverse environmental threat to aquatic life or persons using the water.

The 1981 aerial spraying program commenced in Winnipeg on 4 August and concluded in Roblin on 26 August, 1981. During this period, a total of 21 communities with an area of 455,547 hectares was sprayed with 199,672.4 litres of Baygon. This report provides the results of water analysis for Baygon carried out during the emergency spray program.

METHODS

Baygon monitoring was conducted in 11 of the 21 communities. Water samples were collected at selective sites prior to the aerial application to establish the background levels of Baygon. These sites were resampled after the completion of spraying to determine the concentration of Baygon residues attributed to the actual spraying program. (Table I).

The analytical procedure used for the Baygon analysis in water was developed by the Water Quality Branch of Environment Canada at Calgary, Alberta. The grab samples were collected by staff of Manitoba Environmental Management (MEM). The samples were immediately preserved by adding 10 grams Na_2SO_4 and acidified with H_2SO_4 to pH 3-4 (Anon., 1979).

Baygon was extracted from the water samples immediately after their arrival at the Technical Services Laboratory in Winnipeg. A 1.0 ℓ sample was extracted with methylene chloride, dried over anhydrous sodium sulfate, concentrated and quantitatively transferred to glass ampules. These extracts were then shipped air express to the Water Quality Branch Laboratory in Calgary for the remaining portion of the analysis. The samples were evaporated to dryness, dissolved in hexane and analyzed by gas-liquid chromatography employing a nitrogen phosphorous detector. This resulted in a detection limit of 0.1 µg Baygon/ℓ of water.

RESULTS AND DISCUSSION

Staff of MEM attempted to obtain post-spray water samples up to a maximum of 24 hours after the aerial spray of Baygon. The majority of these post-spray samples were positive for Baygon (Table I). The highest level recorded was in the Boyne River at Carman, Manitoba (i.e., 1.9 µg/ℓ). Because the biodegration of Baygon is apparently quite rapid, particularly in waters with high bacteria levels and at high temperatures the residues detected were low as expected (Bowen, Gavin, Brust, 1976).

The analytical results for the insecticide Baygon in selected surface waters indicated the following, i.e., (a) that drinking water sources were not contaminated with significant levels of Baygon in samples secured up to 24 hours post-spray; (b) that positive detection of residual Baygon in the majority of the post-spray samples collected; and (c) that the Baygon concentrations detected were significantly lower than those detected in a similar program conducted in 1975 in which it was concluded that Baygon residues caused no significant environmental damage and was of no immediate consequence to man.

It would have been beneficial to record the temperature and the pH of the water as Baygon degradation is apparently affected by these factors (Anon., 1982). It is recommended that more concise studies be undertaken in coordination with other environmental monitoring (effects on non-target organisms etc.) to better understand the biological significance of an aerial application of a pesticide such as Baygon.

References

Anon., 1979. Analytical Methods Manual. Inland Waters Directorate Water Quality Branch, Ottawa.

Anon., 1982. Effects of Propoxur on Environmental Quality With Particular Reference to its Use for Control of Biting Flies. National Research Council Publ. NRCC No. 18572.

W. G. Bowen, H. C. R. Gavin, R. A. Brust, 1976. Environmental Monitoring of Insecticide Applications During the Emergency Mosquito Control Operation in Manitoba 1975. Canadian Journal Public Health, 1967 (Supplement): 6 pages.

Table I. Baygon Levels Before and After Spraying in Selected Manitoba Locations During August, 1981.

Sampling Location	Pre-Spray Sample	Baygon (µg/ℓ)	Spray Date	Post-Spray Sample	Baygon (µg/ℓ)
Deacon Reservoir Cell #2 Wpg	Aug. 5	<0.1	Aug. 7	Aug. 8	<0.1
Deacon Reservoir Cell #2 Wpg			Aug. 7	Aug. 10	<0.1
Tache St - Wpg			Aug. 7	Aug. 8	0.2
Tache St - Wpg			Aug. 7	Aug. 10	0.5
Portage la Prairie-WTP[1]-raw[2]	Aug. 5	<0.1	Aug. 11	Aug. 11	<0.1
Portage la Prairie-Assiniboine R	Aug. 5	<0.1	Aug. 11	Aug. 11	0.9
Selkirk - WTP raw	Aug. 5	<0.1	Aug. 11	Aug. 11	0.6
Selkirk - Red River	Aug. 5	<0.1	Aug. 11	Aug. 11	0.5
Morris - WTP raw	Aug. 5	<0.1	Aug. 11	Aug. 12	<0.1
Morris - Red River raw	Aug. 5	<0.1	Aug. 11	Aug. 12	<0.1
Morden - WTP raw	Aug. 5	<0.1	Aug. 13	Aug. 14	<0.1
Morden - L. Minnewaska	Aug. 5	<0.1	Aug. 13	Aug. 14	<0.1
Brandon - WTP raw	Aug. 13	<0.1	Aug. 14	Aug. 14	1.5
Brandon - Assiniboine R	Aug. 13	<0.1	Aug. 14		
Dauphin - WTP raw	Aug. 17	<0.1	Aug. 18	Aug. 18	<0.1
Dauphin - Edwards Creek Reservoir	Aug. 17	<0.1	Aug. 18	Aug. 18	<0.1
Carman - WTP raw	Aug. 18	<0.1	Aug. 20	Aug. 20	1.4
Carman - Boyne River	Aug. 18	<0.1	Aug. 20	Aug. 20	1.9
Neepawa - WTP raw	Aug. 19	<0.1	Aug. 20	Aug. 21	0.6
Neepawa - L. Irwin	Aug. 19	<0.1	Aug. 20	Aug. 21	1.3
Russell - WTP raw	Aug. 25	<0.1	Aug. 24	Aug. 25	1.6
Russell - Reservoir	Aug. 25	<0.1	Aug. 24	Aug. 25	0.8
Killarney - WTP raw	Aug. 25	<0.1	Aug. 25	Aug. 26	0.3
Killarney - L. Killarney	Aug. 25	<0.1	Aug. 25	Aug. 26	0.9

[1] WTP - Water Treatment Plant

[2] Raw - Untreated

A Report on Mosquito Control Programs in Manitoba

S. Eagleton, P. Eng.
Chairman
Manitoba Clean Environment Commission
Bldg. 2, 139 Tuxedo Avenue
Winnipeg, Manitoba
R3C 0V8

The final report, made public on October, 1982, endorsed the Interim Report on Mosquito Control Programs in the Province of Manitoba which was included as Appendix 2. Permission was obtained from the Honourable Mr. J. Cowan, Minister Responsible for Environmental Management, to reproduce the Interim Report in its entirety as follows:

Introduction

This report is a partial response to the request of The Honourable Jay Cowan, the Minister Responsible for Environmental Management, for a detailed investigation of mosquito control programs in Manitoba. This request was made in a letter to the Commission dated March 25, 1982.

This investigation involved four days of hearings in Winnipeg, including two evening sittings, and one day hearings in Brandon, Dauphin, and Thompson. A large number of highly qualified and respected individuals in the fields of medicine, science, academia, Government (Canada and the Provinces), and the public at large, with practical knowledge of matters being examined, made written and oral representations to the Commission, and a correspondingly very large volume of exhibits, papers and transcripts of verbal presentations have been received. The deliberations of the Commission, after thorough study and assessment of all this material, will be the basis for a full and detailed report at a later date.

The investigation carried out by the Commission pertains to two separate aspects of mosquito control:

1. The application of mosquito control in the prevention of an outbreak of Western Equine Encephalitis; and

2. the abatement of the nuisance caused by mosquitoes (nuisance mosquitoes).

The first aspect of mosquito control is a rather urgent matter since the Province may, at any time, be faced with another threat of a Western Equine Encephalitis epidemic. For this reason, the Commission decided to deal with mosquito control as related to Western Equine Encephalitis first and to transmit its preliminary conclusions and recommendations on this matter to the Minister in the form of this interim report.

This course of action will enable the Minister to give consideration to the interim conclusions of the Commission should action be required during the summer of 1982 in connection with a Western Equine Encephalitis prevention program.

Interim Conclusions and Recommendations

The Clean Environment Commission, having considered the evidence presented at the public hearings, has reached the interim conclusion that a program of aerial spraying with the pesticide Baygon is environmentally acceptable at this time, should the Province be faced with a serious threat of an outbreak of Western Equine Encephalitis and should this course of action be recommended by the Manitoba Arbovirus Surveillance Committee.

The Commission also conlcudes that the present practice of having the Manitoba Arbovirus Surveillance Committee monitor the probability of an outbreak of Western Equine Encephalitis and advise the Government on the need for the implementation of an aerial spraying program be continued.

The Commission has based this position on the following considerations:

1. The evidence presented at the hearings, while based on a rather limited monitoring program, nevertheless adequately indicates that the aerial spraying program conducted in 1981 did reduce the numbers of Culex tarsalis mosquitoes, the major carrier of the virus. This supports the conclusion that the 1981 program reduced the incidence of infections with Western Equine Encephalitis virus. No confirmable evidence to the contrary was submitted.

2. The evidence presented at the hearings also indicates that the 1981 single aerial dispersal of Baygon into the environment, with the low concentration used by the aerial spraying program, posed no appreciable threat to human health or to fish and wildlife. Significant damage to nontarget species was reported only for honeybees. There were, however, concerns expressed by some persons at the hearings about adverse long-term effects of repeated chemical applications and the accumulation of any residuals.

3. The Arbovirus Surveillance Program was an effective tool for judging the severity of the threat of a Western Equine Encephalitis epidemic. It constituted an excellent means of bringing together those government officials that have a primary responsibility in this area and a number of experts who need to be consulted. In a multifacetted and uncertain situation, this arrangement appears to be the best and most suitable means of providing the government with responsible professional advice based on a balanced judgement.

Additional Conclusions and Recommendations

Decisions must be based on the best available evidence, even though the evidence is not always complete or conclusive. The Commission recognizes the serious questions and concerns that were raised at the public hearings about the suitability of the aerial spraying program in the control of Western Equine Encephalitis. For this reason, the Commission wishes to present the following additional conclusions and recommendations:

1. The direct evidence for the effectiveness of the aerial spray program in reducing the population of Culex tarsalis, although positive, is based on a limited number of tests. There are also indications that, in some areas, the effectiveness varied significantly from point to point. There is a great need for a aerial spraying is indeed effective and, secondly, to improve the efficacy where possible.

2. An aerial spray program will not eliminate the risk of an infection with Western Equine Encephalitis even in the areas that are covered by the program. In the rural areas that are not sprayed, the risk remains. It is important to realize, therefore, that personal protection against mosquito bites offers the most effective way of reducing the risk anywhere. Previous experience, however, shows that it is difficult to alert and convince the general public of the need for personal protection, even though the measures recommended were rather simple. The Commission, therefore, recommends that methods of increasing public awareness for the need of personal protection be studied and developed and that an intensified program should be a part of any future aerial mosquito control spray program.

3. A vaccine for the control of Western Equine Encephalitis has received attention but with limited success. Unfortunately, this vaccine is not suitable for mass vaccination nor is it suitable for immediate immunization at the time when an epidemice is identified. Neither is it presently available for general use. While it is probable that a vaccine could be developed that would be suitable for mass immunization, it is unlikely that the development would be undertaken by the private sector without further government incentives. The limited distribution of the Western Equine Encephalitis disease and the infrequent occurrence of epidemics discourage private initiative in this area. In view of the seriousness of the disease, the limited protection offered by mosquito control programs and the objections to the aerial application of pesticides, especially in residential areas, the Commission recommends that serious consideration be given to the possibility of encouraging the development of a suitable vaccine by the senior governments.

4. The Federal Government officials, responsible for pesticide registration and testing, present at the hearings gave evidence to the Commis-

sion that Baygon is environmentally acceptable for aerial spraying when used in accordance with authorized dosage directions and application procedures for this purpose. Nevertheless, the possibility exists that exposure to aerial spraying may adversely affect persons in a high risk category, particularly those persons with respiratory diseases and those with an unusual sensitivity to the chemical. The Commission does not consider this sufficient reason for disallowing aerial spraying with the chemical since one can avoid exposure without undue hardship. But the fact that safety cannot be guaranteed for all persons makes it essential that all reasonable steps be taken to ensure that the public is made fully aware of the intention to spray and the timing thereof in each of the areas to be treated.

5. Evidence presented at the hearing indicated that beekeepers suffered losses during the aerial spraying program. Because bees are a significant part of the ecosystem as well as being important from an economic viewpoint, steps should be taken to ensure that the beekeepers considered when developing an aerial spraying program and are then fully informed of the chemical spraying operations.

6. There is a need for the earliest possible identification of "risk possibilities" when Western Equine Encephalitis may threaten. The ongoing program of Arbovirus surveillance, together with the periodic emergency programs, represent a considerable commitment of manpower and funds. The effectiveness of the overall effort is reduced if, during years of a minimal Western Equine Encephalitis threat, the surveillance program is cut for the sake of economy, as has happened in the past. In addition, there are still unanswered questions about the epidemiology of Western Equine Encephalitis and the effect of the effort to control the disease. To maximize the effect of the Western Equine Encephalitis control/prevention program, it is necessary that such uncertainties be reduced as much as possible. The Commission, therefore, recommends that the surveillance program be provided with stable funding so as to obtain data that is consistent from year to year.

   The Commission furthermore recommends the funding of research into the annual cycle of the virus and into the effectiveness of the current program in modifying the natural course of an epidemic.

CHAPTER IX:

Human Protection

THE ROLE OF PERSONAL PROTECTION IN THE PREVENTION OF WESTERN EQUINE ENCEPHALITIS

D.G. Luckhurst, B.Sc.,M.N.R.M.,M.D.
Deputy Medical Health Officer
City of Winnipeg Health Department
280 William Avenue
Winnipeg, Manitoba
R3B 1B9

J.A. Eadie, M.B.,Ch.B.,D.P.H.
Provincial Epidemiologist
Preventive Medical Services
Manitoba Department of Health
831 Portage Avenue
Winnipeg, Manitoba
R3G 0N6

Abstract

Strategies for primary prevention of Western Encephalomyelitis when monitoring parameters indicate an incipient human outbreak, are few in number. The role of public education campaigns advocating minimization of individual exposure is discussed.

Preventive health monitoring programs must, by definition, have as an integral component an intervention strategy, ideally one which is both acceptable and efficacious (Whitby, 1974). Although the Manitoba Arbovirus Surveillance Committee (MASC) has, through its monitoring efforts, uncovered information of a research nature on the biological basis of this disease (Sekla, 1976), the work of the Committee is viewed by policymakers primarily as a preventive health program (Anon., 1982 a). Hence, the continued support of its work is contingent on its capability to invoke, when appropriate, effective and efficient means to prevent human disease (Anon., 1982 b).

However, the identification of an incipient outbreak of Western Equine Encephalitis (WEE) prevents few alternatives for preventive action. Large scale aerial adulticiding has been the main strategy invoked for all three recent epidemics, although concerns regarding efficacy (Anon., 1981 a) and, more particularly, acceptability (Anon., 1981 b) have been raised frequently.

In the absence of a practical vaccination program for WEE, the only alternative preventive tool is advocacy of personal protection through a publicity campaign. However, placing greatest emphasis on this latter prevention strategy and reducing or excluding the spray approach has not been deemed wise based on its failure when attempted during earlier epidemics.

In the epidemic years of 1975, 1977, and 1981, repeated announcements to the public drew attention to such measures as the following:

(a) wearing light-coloured clothing, with long pants and long sleeves to reduce the amount of exposed skin;
(b) minimizing out-of-doors activity during the emergency, particularly at the sunset hour;
(c) liberal use of mosquito repellants;

(d) apply all the measures for personal protection especially to children and elderly people since some members of these age groups are unable to recognize the need, nor take action themselves; and

(e) keeping screen doors and windows in good condition (Anon., 1981 c).

The preventive measures suggested were sound and, if widely observed, an impact on prevalence could be expected. This is particularly so with regard to insect repellants since virtually all of the numerous commercial preparations (most containing diethyl toluamide or ethyl hexanediol) are effective for at least 2 hours (Gabel, 1976) and pose little risk of adverse reaction unless used excessively on small children (Anon., 1968).

Nevertheless, compliance with these recommendations was deemed to be low by MASC. In part, this judgement was based on reviews of compliance with other preventive health recommendations, officially promulgated, in programs wherein the risk of severe adverse health effects was similarly remote. Thus, in antihypertensive, weight reduction, anti-smoking or drinking moderation programs, very often the risk assessment tests are good, but resulting compliance makes the effort questionable (Holland, 1974).

Perhaps of greater weight, however, was an anecdotal experience of some MASC members. On attending outdoor events, for example, in the risk area, as the sun set, with calm, warm conditions, one would note colleagues in shorts and tee-shirts with no repellant on, while the MASC member wore long sleeves and pants and emitted a heavy odour of bug spray.

Personal experiences notwithstanding, MASC recognized, on review of the 1981 outbreak experience, that much more effort in this potentially fertile area is required. For one thing the scale of the public information program has been compatible with the objective inherent in its title: information dispensing. This is the view of other mosquito control programs as well (Anon., 1978 a) but it was felt that a redefinition of the objective of the publicity program was required so that education, with a view to alteration of health behaviour, would be the accepted goal. Staged with events as they develop over the summer, the scale of such a campaign could be dramatically increased by mail inserts, mall displays, television documentaries, and paid advertisements. A plan is being prepared, under the auspices of MASC by health education specialists, to develop a detailed promotion campaign, graded according to risk of outbreak and including these as well as other outreach techniques.

Since efficacy may well be dependent on the scale of the program, a more scientific assessment of the value of a personal protection campaign as a public health intervention strategy will be possible only when evaluated at different levels of effort. Although studies of efficacy can be difficult, some methodologies exist and have been attempted in this province (Anon., 1978 b). Recognizing that the acceptability of spraying is a continuing emotional issue, the vigorous pursuit of personal protection campaigns is viewed as a reasonable, if not difficult, alternative.

While no studies were made of the use of repellants during the emergency, personal and anecdotal observations suggested that a majority of the population took neither heed nor action to minimize their personal exposure to mosquito bites.

Although farmers appear to be the highest risk group, it would appear that they, after many years of exposure to mosquito biting, develop a "tolerance" to bites and see little need for personal protection.

References Cited

Anon., 1982 a. Encephalitis control two-day workshop set. Govt. of Manitoba News Service. February 12, 1982.

Anon., 1982 b. Report says encephalitis caught province off guard. Winnipeg Free Press. February 18, 1982.

Anon., 1981 a. Concerns raised about spraying. Winnipeg Free Press. August 1, 1981.

Anon., 1981 b. Use of sprays to kill pests questioned. Winnipeg Free Press. June 22, 1981.

Anon., 1981 c. Mosquito war gets under way again. Winnipeg Free Press. June 22, 1981.

Anon., 1978. Control of Western Equine Encephalitis. USHEW Vector Topics No. 3. 35 pp.

Anon., 1968. Insect repellants. Medical Letter 14: 55-56.

Gabel, M.L. et al, 1976. Evaporation rates and protection times of mosquito repellants. Mosquito News 36: 141-146.

Holland, W.W., 1974. Screening for disease: taking stock. Lancet 2: 1491 - 1497.

Robertson, D. 1978. Report on Venereal Diseases Public Awareness Campaign, 1978. Manitoba Department of Health. 26 pp.

Sekla, L.H. (Ed.) 1976. Western Encephalomyelitis. Can. J. Public Health 67 (Suppl.) 75 pp.

Whitby, L.G., 1974. Screening for disease: definition and criteria. Lancet 2: 819-822.

[illegible]

[illegible]

[illegible]

The Case for Control of WEE in Humans Through Vaccination

J.G. Fox M.D., FRCP(C), Dip.Bact(Tor.)

Manitoba Health Services Commission
Cadham Provincial Laboratory 750 William Avenue
Winnipeg, Manitoba R3C 3Y1

Abstract

Vaccination has been a control mechanism for WEE in horses for many years. Its use in humans has been largely limited to field workers and laboratorians investigating the disease. Control of episodic outbreaks of WEE in endemic areas by vaccination of the population at risk would offer many advantages over the present method of reducing the vector mosquito population by application of chemicals. However, vaccines produced to date for human use are not sufficiently antigenic to produce long term immunity. The production of a vaccine that would induce rapid and long term immunity after a single dose would require considerable research and development and would be very costly as there is only a small endemic population base that might support it. For the immediate future at least, a vaccine does not offer a viable alternative to existing methods of control

Although a vaccine for WEE has been produced in limited quantities for certain select groups such as members of the U.S. Armed Services, field personnel in public health and laboratory workers, there is no commercially available vaccine today for massive population immunization.

Toward this end, a vaccine study project was undertaken by Saskatchewan in 1971 with the support of a grant from Health and Welfare Canada and in conjunction with Connaught Medical Research Laboratories. Methods of growing the virus and making a vaccine were studied. A formalin inactivated vaccine prepared from virus fluid grown in chick embryo cell culture was produced and subjected to limited field trials.

POSSIBLE ADVANTAGES OF A VACCINE AS A CONTROL MEASURE

a. Elimination of the need for widespread geographic aerial spraying of pesticides and hence the possible threat of toxicity to man and his environment attributable by some to chemicals of any kind.

b. The seemingly better use of funds directed towards producing a permanently immune population compared to the episodic elimination (often incomplete) of a single generation of adult mosquitoes in a single season.

c. Elimination of the need for maintaining/acquiring year after year the necessary logistic and support capability required to make spraying an effective emergency modality, taking into consideration the irregularity and infrequency of the indication for its application.

d. Reducing or even eliminating the need for annual surveillance procedures as widespread immunization and resultant population immunity reduced human morbidity and mortality to acceptable limits.

e. A suitable vaccine would provide whole season and multiseason protection in contract to insecticides which has only a short term effect and consequently allows for early or late season infections to occur depending on the timing of its implementation.

f. A suitable vaccine would be expected to provide a statistically greater degree of protection to the individual compared to pesticiding where 40 to 70% reported success rates in vector kills are usual, depending upon the topography and other variables in conditions existing during the spraying. Local experience has shown that the implementation of large scale insecticiding often delayed beyond the point of maximum effectiveness in many locales, although earlier intervention, if indicated, might be expected to result in the need for repeat application (s).

g. The provision of immunity through vaccination would allow the protected individual the freedom of moving about the province without risk, in contrast to the limited geographic and temporal risk control associated with a spraying program timetable.

h. Insecticide control might be expected to lead to the build up of resistant mosquito species.

i. A vaccine would also be valuable for the protection of high risk groups such as serology and virology laboratory workers.

LIMITATIONS OF THE APPROACH TO WEE CONTROL BY HUMAN VACCINE ONLY

a. Mosquito species not known to be vectors of encephalitis viruses will still require control measures to be put in place when their numbers exceed a certain threshold level of nuisance tolerance in the community. Accordingly, larviciding programs, residential insecticide fogging and the personal use of repellents will continue regardless of a vaccine program.

b. In view of (a) above, it could be that aerial spraying would be the only component of mosquito control that a vaccine program would make redundant. If this is the case, the ideal solution to both the nuisance and disease vector problem associated with the high mosquito counts would appear to lie in researching a safe, non-chemical (biological) method of mosquito control.

c. The employment of a vaccine, should a suitable one be produced, would still require a good deal of judgement and consideration in its use, even in an endemic area such as Manitoba. This is based on certain known and some incompletely known epidemiologic and pathogenic features of the disease. Some of them are:

1. The irregularity of human epidemics.
2. The potential risk of clinical illness that exists for most of the population in the face of a very low attack rate even in "epidemic years".
3. An incompletely studied, but probably low exposure rate of around 5% judging by a previous serologic study in Manitoba.
4. No assembled epidemiologic data from the different regions of the province from which to calculate the ratios of severe illness/mild illness/subclinical infections.
5. Non-existing, up-to-date, hard data on the percentage of the population with significant levels of neutralizing antibody by age, sex and region.
6. No previous investigation for the presence of other possible host factors other than antibody studies, which might explain the variability of clinical manifestations in different individuals, (e.g., Cellular Mediated Immunity).
7. No studies on the possible variability in neurovirulence of isolates from different regions, hosts, different years.
8. The logistics of, the acceptability and anticipated probable compliance with, a mass program should it be indicated.
9. The possibility of antigenic variation in isolates from different regions, hosts, years. No studies correlating antibody levels with protection against encephalitis.
10. The necessity or desirability of tests for immunity prior to vaccine administration.

d. The disease cycle in nature is left untouched by a human vaccine.

Despite earlier work on vaccine production by Connaught Medical Research Laboratories and field studies with different lots of vaccine conducted by Saskatchewan, a completely suitable safe and effective prototype vaccine for mass immunization of a population is still not available. A perusal of the literature does not indicate that one is available elsewhere in North America either. Although some good and valuable pioneering work towards producing such a vaccine has already taken place in Canada, much expensive and time-consuming research and development together with all the necessary field trials remains to be done before a vaccine could otherwise be considered an option. The long term effects i.e., disease prevention v.s. antibody persistence and remote side effects would take several decades to assess.

NECESSARY CHARACTERISTICS OF A HUMAN VACCINE FOR POPULATION PROTECTION

The incidence of serious illness and complications on a population wide basis associated with WEE is low relative to the risk attending to most other infectious diseases where the development of a vaccine was considered at the time of introduction to be the ideal method of control. It is important, therefore, that a WEE vaccine have certain features which might not be considered crucial in another vaccine. Adding importance to this is the probability that a determined effort by the individual citizen to avoid unnecessary outdoor exposure can reduce the possibility of infection close to extinction. Accordingly, in addition to the usual required properties of a vaccine relating to antigenicity, protection, toxicity, etc., that must pertain before licensing for the use of a WEE vaccine should, have at least the following properties:

1. The ability to provide rapid protection following a single inoculation. The Saskatchewan vaccine did not meet this requirement. The ability to produce a rapid sero-conversion would be important in instances where the administration of the vaccine was made in the face of an impending epidemic.

2. Life-long protection against clinical encephalitis. It would clearly be impractical to revaccinate using a multiple dose schedule or even to administer a single dose booster to large numbers of the population if the initial immunization series did not provide solid immunity for at least a reasonable time period (e.g., ten years).

3. The ability of the vaccine to maintain its potency after stockpiling for a number of years. This attribute in a "one dose" vaccine would enable its use to be deferred until an impending epidemic appeared likely should this strategy for its use be considered.

4. Compatibility for combination with other immunizing agents. Simultaneous administration in combination with other childhood immunizing agents would simplify the administration of the vaccine to the infant population. Administration at this age would, however, underscore the importance of the vaccine having the ability to stimulate protective antibody of long term duration.

5. No serious immediate or delayed side effects (e.g; local and systemic reactions).

SUMMARY

Vaccination as a method for control of WEE in Manitoba would have as its chief advantage the elimination of the need for episodic chemical aerial spraying of large areas of the province when an outbreak appeared to be imminent, as is the situation at present. It would also be expected to modify the need for an annual monitoring program and to provide all season protection to the community as opposed to post spraying control.

However, at the present time, no vaccine exists that is suitable for mass immunization of an entire population in endemic areas despite efforts to produce one in Saskatchewan and the U.S. in the last decade.

The factors mitigating against a vaccine as a control measure include at least the following:

1. There does not appear to be an easily identifiable high risk subgroup so that vaccine would need to be administered to the entire population - a horrendous logistical undertaking.

2. A vaccine would need to be developed which had qualities not hither to obtained in earlier attempts, i.e. high antigenicity, long term protection, long term stability, single dosage. Development of such a vaccine would take years for its safety and protective capabilities to be fully evaluated. The sizable costs involved would likely have to be borne entirely by Manitoba as there appears to be little interest elsewhere at the present.

3. The periodicity and sparcity of infections in the population and the relatively low morbidity and mortality in those infected is an unfamiliar scenario for control through vaccine development and application

For the near future at least, control of WEE in Manitoba by immunization does not present a viable alternative. Nonetheless, as new technologies in the development of viral vaccines evolve, it is possible that vaccine control could yet play a contributing role in the resolution of the problem in Manitoba.

CHAPTER X:

Comments Made At The 1982 Western Equine Encephalitis Workshop on Manitoba's Arbovirus Surveillance and Control Programs

Comments on Manitoba's Arbovirus Surveillance Program

D.B. Francy Ph.D.

Vector Borne Disease Division
Center for Disease Control
U.S. Public Health Services
Box 2087
Fort Collins, Colorado 80522

I would like first of all to take this opportunity to express my appreciation for being invited to participate in your meeting. It has been enjoyable and informative for me and I hope that it will continue. Before I get into any suggestions or comments, I would like to say that, as a result of the presentations yesterday and what we have heard of the surveillance program and the efforts to deal with the problem of Western Equine Encephalitis (WEE) virus as a public health problem, overall I am quite impressed with the job done in Manitoba. I think this public health problem has been addressed in a very responsible fashion. The work that has been done is of high quality. The Manitoba Arbovirus Surveillance Committee (MASC) has applied most of the methodology that is available that has worked in other areas and they are doing a really excellent job.

As with any sort of program there are probably opportunities to modify or institute various techniques. Perhaps an important aspect of all this is that because many of us are involved with surveillance in different parts of the world or country, we would like to see, as much as possible, that some degree of standardization of indices are generated and used for predictive purposes. With this in mind, I think I will start with some comments or suggestions related to mosquito vectors.

I have the impression that the problem with WEE virus in this area is related more to the natural conditions (i.e., flooding of native or prairie grassland, sloughs, and similar habitats) rather than irrigated agriculture. I think, therefore, that it is safe to presume that this is a problem that is going to be with Manitobans for some time to come and is not likely to go away. It seems quite reasonable to continue to implement and support surveillance programs. There has to be a fairly solid financial base for achieving continuity of the surveillance program which means that the necessary funds are being provided to get the work done. It is highly desirable to minimize the amount of fluctuation in financial support that occurs from year to year. It is usually quite easy to generate money for surwillance work the year following a large epidemic but, each year that you move away from an outbreak year, it becomes increasingly more difficult to generate support for these programs. If there is ever to be an opportunity to generate data over a relatively long period and to be able to utilize these indices for predictive purposes with a high degree of confidence, it is necessary to adequately support these kind of programs. Surveillance programs cannot be operated without personnel, equipment and supplies, and laboratory backup.

One of the drawbacks that I feel we have had so far is that we have not had an opportunity to sit down with some of the people who presented information yesterday and discuss with them what their resources are in terms of personnel; what their plans are, etc. Some of my comments may then come out of ignorance of what all the individuals involved have planned for in the future. Since the problem we are dealing with here is to establish workable surveillance systems, one of my suggestions would be to keep in mind, as much as possible, that the surveillance system itself be separated from any sort of research endeavours. I am not saying research on some of the obvious problems is not important, but that those techniques that are judged most useful for predicting levels of virus activity should be the ones that receive major attention. Every effort should be made to shore these up, not only in terms of their representative for the area geographically but also to enhance the efficiency or speed with which information is generated.

There is a great deal of variation in the way information on mosquito populations are expressed. For instance, in the discussions so far most of the data pertaining to mosquito populations has been expressed as total mosquito collections and the percent of that total which is Culex tarsalis. One way that this data might be expressed in a somewhat more straightforward fashion might be to deal just with C. tarsalis because that species is, in fact, the mosquito that we are most concerned with. This data should be expressed as an index of C. tarsalis on a per trap night basis. If you collect a large number of mosquitoes and 10% of those are C. tarsalis, you are talking about a lot more mosquitoes than if you collect a small number of total mosquitoes, yet the proportion of C. tarsalis is still 10%. It would be easier to follow and more meaningful if there was a standardized way of expressing population indices. Primary emphasis should be on C. tarsalis since that is the important species as far as WEE transmission is concerned.

Similarly, it would be somewhat easier to compare seasonal and annual changed in virus infection rates in mosquitoes by examining minimal infection rates (MIR) rather than the percent of positive pools. Usually, minimal infection rates come very close to the actual infection rate in the mosquito pool. If you are dealing with relatively large mosquito pools (e.g., 50-100 mosquitoes) you have to approach 80 - 90% positive before you significantly depart from the real infection rate. The MIR is simply calculated by taking the number of virus isolations (i.e., positive pools) and dividing by the total number of mosquitoes tested and standardizing that by multiplying by a thousand. This is a convention that is used by a number of workers. It is fairly straightforward, and simplified the comparison of infection in mosquitoes on a seasonal basis.

A major effort should be made to collect and test C. tarsalis mosquitoes on a current basis. We all hate to see information thrown away by not testing other mosquito species, but I think that for the surveillance program some compromises need to be made in this regard. The other mosquito species can be frozen and tested at a later date without any concern for loss of viruses, such as Cache Valley or Hart Park or whatever agents one might be interested in. The major emphasis should be on collecting and testing C. tarsalis to form a representative sample.

There seems to be many advantages to the use of bait trap collections. One is that they selectively collect more C. tarsalis than other techniques such as light traps. The other consideration is that, from data that Dr. Brust presented, the bait traps seem to sample C. tarsalis somewhat earlier. That certainly is an advantage. More emphasis can be placed on the bait trap collections. They have worked well in some areas and in some areas they have not. They have the advantage of being highly portable and you do not have the expense involved with the long-term maintenance of sentinel flocks. That is not meant to be a criticism of sentinel flocks because they are very useful under certain circumstances. Bait traps are better from the standpoint of portability. They can be baited with either a chicken or dry ice. They would be useful in expanding the sampling of live C. tarsalis for virus studies. It appears to me that C. tarsalis mosquito populations in this area in most years are not tremendously abundant and that the use of CDC light traps supplemented with dry ice might be a useful tool for sampling populations, not only for population indices but also for virus isolation. They have the big advantage over New Jersey light traps in that you are not limited to a source of electrical power: they are portable, run on flashlight batteries and collect a lot less of other insects such as beetles and moths. Field workers are highly mobile when using these traps. If you have a special interest in an area as a result of data that is developed during the season, you can go to that area and supplement your normal collections to increase the data available.

There are certainly no disadvantages at all to submitting live mosquitoes to the laboratory for testing from the standpoint of virus isolation. That is an optimal way to provide mosquitoes for testing but I think, if it means creating problems for the field people or for laboratory people in handling these specimens, then there is no problem freezing specimens on dry ice and holding them until they are thawed, identified on a chill table of some sort, and, alternately, then can be identified, pooled and then frozen for subsequent laboratory testing. WEE virus, in our experience, has always been a very hardy "bug" if it is kept at dry ice temperatures, or lower, in a mechanical freezer. There is little chance of losing virus in the mosquitoes prior to the time they are tested.

Any time that procedures are changed or modified, you need to insure that you have some degree of continuity to your indices. If you incorporate different trapping systems such as bait can traps or CDC light traps, you will need to run them for a period of time with other previously used techniques to develop a comparability index. Then, you will not be suddenly dropping one thing in favour of something else with no idea of what the new system means relative to the kind of data that you generated in the past. I think one needs to be very careful about comparability and continuity and that comments should apply to any of my suggestions for changes or modification of techniques. I think that you could make good use of chill tables or some other sort of cooling device for sorting mosquitoes. It is a very laborious procedure to catch mosquitoes in individual vials for identification. I think they can be knocked down with a little CO_2 or be chilled to the point where they are knocked down. They can be emptied out into Petri dishes kept on chill tables where they are readily identified. I think it would be a good investment to consider getting something like this for working with mosquitoes.

Another issue, but one which I think is important as far as the vectors is concerned, is the possible modification of some of the laboratory procedures used for virus isolation. In our experience, duck embryo is a very sensitive assay system for WEE virus. If it is possible to obtain and prepare the duck embryo it would certainly be good to evaluate. Plaque assay, using an overlay of agar-nutrient media containing a vital stain for visualizing plaques, is a less labor intensive way of looking for virus and may, in fact, be somewhat more sensitive than looking at Cytopathic Effect (CPE). The crucial thing for surveillance is rapid isolation and identification. After you have used the plaque assay procedure for awhile, you can identify isolates with a high degree of probability based on plaque morphology and incubation time. WEE virus plaques usually come up 24-48 hours after they are inoculated and there will be little doubt in your mind that you have a WEE virus isolate.

However, for confirmation or identification, there are several rapid techniques for identification. One would be passage of the harvest material into cell culture grown in lab-TEK chamber slides for direct Fluorescent Antibody (FA) staining. The other procedure used is to inoculate 2 cell culture flasks, 1 with neutral red in the overlay and 1 without. When plaques appear, remove the agar from the unstained bottle, add borate saline and place the flask into the incubator overnight at 4°C. Remove the borate saline which then serves as a hemagglutinin. This is diluted in the presence of known immune sera, diluted to eliminate cross reaction with closely related viruses. You can thus make a very rapid isolation and identification of virus. I think that generating information as quickly as possible for the use of the surveillance committee is the desired goal. I would discourage as much as possible the combining of mosquito pools for inoculation. That just makes additional dilutions of the specimens when you combine pools and I think may decrease the chance of isolating viruses.

I think it would be desirable to do some studies on the mosquito population over time. This provides an indirect way of estimating what proportion of the population may be able to transmit virus. Beside the sampling procedures already mentioned, other possibilities for collecting mosquitoes, the removal of mosquitoes from resting sites or using truck traps. Truck traps are relatively unbiased and collect those mosquitoes flying at a particular time. They have the disadvantage of being labor intensive and you have to have people running the traps at the appropriate time of day or early evening. In the same regard, I think that this sort of sampling procedure is very useful for pre- and post-spray evaluations. I think that it would be desirable to "shore up" that work considerably, relative to your control program.

I do not know what the turn-around time is, in terms of getting results from testing sentinel chicken sera to the surveillance committee. Again, it is certainly desirable to reduce the time that it takes to get the specimens to the laboratory and information back to MASC as much as possible. I would like to mention at least one procedure that worked in some of our studies in West Texas. I am not suggesting that this takes place of sentinel chickens but, at one time in West Texas, we put out sentinel hamsters because we were mainly interested in detecting WEE virus activity. A number of hamsters died very suddenly. The person that was in the field at the time discarded these specimens because he thought they were dying from the heat. We finally obtained a few of them and found that the brain of each dead hamster was loaded with WEE virus. It seems that at least in that area, hamsters were very susceptible to peripheral inoculation of WEE virus by mosquitoes. This might provide a technique in which you would not have to worry about the secondary testing of chicken sera. You might try to incorporate that, along with 1 or 2 sentinel flocks to see if, perhaps, it would work in this area. It is easy to identify virus in the brain material by FA and this might be a useful procedure. One drawback might be that *C. tarsalis* feeds primarily on avians early in the season, at least in the U.S., and that might decrease the usefulness of a mammalian sentinel.

Larval indices are something that we really did not hear anything about and I am sure that there is a great deal of information available from the City of Winnipeg's Mosquito Control Program. If systematic, larval survey work is done, I think that these indices could be used to anticipate adult populations. They are less subject to day-to-day weather variations that greatly affect adult mosquito collections. These also might provide the grist for the modelling which although I do not really understand all that well, might allow you to predict what is going to happen. Certainly, if you can do that a month or two weeks ahead, it would be desirable even if you are sometimes wrong. It does provide additional information for consideration, I think any information that can be generated is useful.

I would like to see some work done with the wild bird population, perhaps initially on a survey basis looking for antibody prevalence. Try to zero in on perhaps one, two or three species of birds that seem to be particularly involved as maintenance and amplifying hosts. It appears to me that WEE virus is endemic in this area. There must be some species that are more involved than others as far as birds are concerned. Once you identify what these avian hosts are, it may be possible to try to work towards sampling nestling populations for virus isolation as a very useful indicator of what is in store as far as WEE virus activity is concerned. Virus isolation from nestling house sparrows proved to be a very sensitive and useful index of WEE virus transmission in West Texas studies.

Along the lines of research needs, I think there is a great deal of interest in Culiseta inornata as a possible early season vector of WEE virus. As a research project, aside from the surveillance program, it would be extremely interesting to save all engorged specimens of this species for bloodmeal identification to determine the cross feeding range of this species. That might provide a clue as to which animals might be involved in the virus cycle. If, in fact, C. inornata females do feed a great deal on birds, then that would be a situation fairly unique to this area. We see a great deal of variation within a species from area to area in terms of feeding preferences so I would not be terribly suprised if these mosquitoes do feed readily on birds.

In response to a question about a technique to correlate the percentage of trapped mosquitoes with their ability to transmit infections, I replied that the only operational technique that I am aware is the one that was applied by Dr. W. Reeves, in California, a number of years ago. They used bait cans and exposed chicks in these bait cans to the mosquitoes that were being trapped. The mosquitoes feed on the chicks when they come into the trap. Testing the exposed chicks for evidence of infections, as well as testing the mosquitoes for virus provides a rough index of the proportion of infected mosquitoes capable of transmitting virus. In fact it provides a fairly useful index in the field and I would suggest that anyone that is interested in this refer to Dr. Reeves' paper.

In response to a question concerning sporadic cases of St. Louis Encephalitis (SLE), I replied, that it is our collective view, as far as SLE virus is concerned, that it tends to be active in more Southern or warmer regions. This is partly because the virus has a longer generation time for replication in vectors. In an area like this, with a relatively short and intensive mosquito season, the likelihood of an intense epidemic transmission is not too great. A SLE epidemic is very unlikely.

In reply to a question on the value of aerial spraying from the floor, I said: "I can appreciate the concern". You are faced with a repetitive situation where every other year or so you make a choice to put 2 or 3 million dollars when you do not know whether aerial spraying is doing what you want it to do. Some of the suggestions I made here in terms of using non-human indicators of virus activity may be one way of getting around the problem. Faced with the indicators of virus activity that were present in 1981, I think it probably almost would border on public malpractice not to do something to protect the population.

In reply to a question on the value of aerial spraying [illegible] the [illegible], I said: "I can't calculate the [illegible]. You are dealing with a repetitive situation and every other year or so you have a choice [illegible] use [illegible] dollars [illegible] the people have whether aerial spraying is going [illegible] and want it to do. Some of the suggestions I have here in large [illegible] indicators of virus activity may be one way of getting ahead [illegible] in 1969, I think it is worth a great deal of [illegible] in making [illegible]

[illegible]

[illegible]

[illegible] that was applied by Dr. [illegible] Reeves, in California, a number of [illegible] have been used [illegible] that were being [illegible] selections, as well as [illegible] index of the presence of an [illegible] encephalitis virus. In fact it provides a [illegible] index to the field and I would suggest that [illegible] be incorporated in this [illegible] period.

In response to a question concerning Japanese B and St. Louis Encephalitis (SLE), I replied, that [illegible] the vector side, as far as the virus is concerned, that it seems to be milder in more Southern or warmer regions. This is partly because the virus has a longer generation time for replication in vectors. In an area like this, with a relatively short and intensive mosquito season, the likelihood of an intense epidemic transmission is not too great. A [illegible] epidemic is very unlikely.

Comments Made at Manitoba's Western Equine Encephalitis Symposium

T. Monath M.D.
Director

Division of Vector-Borne Viral Diseases
Center for Disease Control
P.O. Box 2187
Fort Collins, Colorado USA 81522

I would like to congratulate the Manitoba Arbovirus Surveillance Committee (MASC) for having organized and implemented what I consider to be a really first-class surveillance program for Western Equine Encephalitis (WEE). The critical elements of this program include the gathering of information on vector density, the collection and testing of specimens for virus isolation, the determination of virus transmission to sentinel flocks, the application of good laboratory testing, the frequent evaluation of data generated by MASC, the use of these data for making decisions, and finally, the implementation of emergency vector control with a lot of advance planning and minimal possible delays. This sort of an integrated and coordinated approach to the problem of mosquito-borne encephalitis is exactly what is needed. I think this a model system, and I wish that we, in the United States, were facing the problem of arbovirus surveillance as adequately as you are here.

I am going to make some general comments on the problem of surveillance, then go on specifically to some aspects of the epidemiology of WEE, and finally comment on the important question of how to assess the efficacy of control. Dr. Brust presented a very clear picture of what appears to be a strong correlation between *Culex tarsalis* and vector density, measured best using the chicken-baited traps, and the occurence of human disease. The data has been derived by observations over a five year period. In epidemic years, there must be a sufficient vector population. The parameters that appear to warn of an epidemic are the proportion of the total mosquito population (over 25%) made up by *Cx tarsalis*, which is trapped at a rate of over 20 - 40 females per trap per week in the chicken baited traps before mid-July. These data were impressive. I think it is clear that *Cx tarsalis* abundance, as defined by these indices, should continue to be a major focus for MASC for future decision-making. However, I think we must recognize and keep in mind the limitations of this particular parameter if used alone. You need to combine it, as you do, with other indices. There are some rather notorious examples in the United States in localities in which *Cx tarsalis* vector density has been used alone as an early warning parameter. High *Cx tarsalis* population densities were found in years without virus activity whatsoever, for example, in California in 1969. So far in Manitoba, of course, the correlation looks very good between vector density and disease but this may not always be the case. You are also looking directly at virus activity, expressed as infection rates in *Cx tarsalis*.

This parameter adds significantly to the usefulness of your data. The data that Dr. Sekla presented seemed to clearly show that the appearance of virus in Cx tarsalis at the time of the rapid increase in vector population in early July had a predictive value. The parameter that was used is the percentage of positive pools, and 2% seemed to be the magic number by mid-July. Here again, I would like to emphasize the need to standardize the way these data on virus activity are used. Analysis of virus infection in terms of minimum infection rates (MIR) obviates the problem of variation in vector pool size. It would be useful to go back and reanalyze your weekly or bi-weekly data on the basis of Cx tarsalis infection rates. The addition of vector infection rate as a surveillance or predictive measurement will be very useful. However, I would also like to point out that there have been some well-documented cases of both high Cx tarsalis population densities and high WEE virus infection rates (based on testing mosquitoes for virus) without the occurence of human disease. I would refer you to a paper by Dr. Reeves, in the American Journal of Hygiene, Volume 80, page 205, 1964. In this situation, vector density and virus infection rates did not correlate with a high rate of transmission. There are a variety of possibilities why this might be true.

A second point with regard to the surveillance of vectors for virus isolation is the possible need to reassess the sensitivity of your present assay systems and to examine ways of speeding up the procedure between the receipt of specimens and isolation and identification of WEE virus. Sensitivity in the assay system is absolutely critical especially early in the year.

The sentinel flock program adds a third dimension to the surveillance and is a parameter that most directly measures viral transmission. Transmission is the element missing from the previous 2 surveillance techniques (i.e., vector abundance and infection rate) and I wholeheartedly endorse the sentinel flock program. I think that because of the question raised earlier on St. Louis Encephalitis (SLE) virus, the sentinel flock sera should be tested both for SLE and WEE antibodies. Because of the large area of Southern Manitoba that needs to be placed under surveillance and the logistics of sampling wild birds for antibody or nestlings for viremia, I think that surveillance of these hosts is not practical.

Human and equine surveillance is an area that probably deserves a little harder look than it has been given in the past. Obviously, you are using equine disease reporting in your decision-making process and it is true that usually equine diseases have preceeded human cases by 2 to 3 weeks. However, unless I am wrong, looking at the epidemic curve last year, the earliest equine and human cases had their onset of illness in the same week in mid-July but the human case was not recognized until much later because the diagnostic specimens were not available until mid-August. I would call your present system of equine surveillance and certainly the system of human surveillance, a semi-passive or semi-active one. Veterinarians have been advised to submit specimens and information if they see a sick horse. Similarily, human practitioners in the Province are being advised of the risk of enecephalitis and are told what specimens to submit. However, it is not a truly active surveillance system in the sense that someone telephones on a regular basis, a sample of the veterinarians of physicians to gather active information and keep the level of surveillance up. In 1981, a month elapsed between the onset of the first human case and recognition of that case. If information on human cases had been available to you at the time you were making decisions on vector control, those decisions would have probably been a lot easier. Thus, a truly active program of human case surveillance would be very advantageous.

Also, during the course of an outbreak, it may be useful to alter the case definition somewhat from the restrictive one of a central nervous system infection to a broader one including non-descript illness with a fever and headache in order to extend the human surveillance system even further. I was impressed with the efforts being made in the laboratory regarding human diagnosis.

Dr. Friesen told us of his detailed analysis of 24 human cases in 1981. A number of the epidemiological features of these cases fit the observations that we are all familiar with from previous outbreaks of this disease: namely, excessive cases among males and high attack rate in farmers and rural residents. Infants and your children and the elderly are known to be more highly susceptible to severe clinical disease but there was only one case in these age groups in this past year. The absence of cases in infants suggested to some that the efforts made to increase public awareness to avoid mosquito bites, which may have been heeded more by mothers of small children, may have been a factor. I certainly endorse those efforts to increase public awareness.

I perceive a real need for a well designed, randomized, stratified serologic survey in Manitoba. Evidently there has not been one done for a number of years. I think this could provide information on the incidence of inapparent infections in recent epidemics, although I doubt it would tell you whether they occurred in 1975, 1977 or 1981. A serosurvey would give you information about age specific inapparent to apparent infection rate. I am not happy with the figures that are published and I think this needs re-examination. Most important, a survey would provide some idea of the relative risk of acquiring WEE infection by geographic area in Manitoba and thereby provide useful information for planning surveillance and control activities. Finally, it would also give you some information about SLE virus activity. I would remind you that interpreting serosurvey data is a little more difficult than with some other arboviruses. The studies that were reported by Froeschle and Reeves in 1960 clearly show that WEE antibody probably persists for a shorter length of time than antibodies to other arboviruses. This creates some problems with interpretation.

Dr. Friesen spent a lot of time examining factors that were associated with human cases in Manitoba, such as time spent out-of-doors, animals nearby, mosquito breeding sites and so on. I found these data almost impossible to interpret, mainly because of the lack of any controls for comparison. The classical approach of the case control study might be applicable here. I will not go into the details about how this may be done but I think it would provide one approach to answer these kinds of questions.

Finally the $64,000.00 question, which will undoubtedly come up again: i.e., "How efficacacious were the mosquito control efforts?" Efficacy of control has not been assessed in terms of human and equine morbidity. From the data presented, it appears to me that WEE virus transmission, to both horses and man, occurred in the Province as significant rates long after the completion of the aerial spraying. One has to conclude that there was a transmission to horses and man after aerial spraying, given the usual incubation period. I think the major reason for this is probably that the cases that occurred after spraying resided in areas that were not sprayed, these being predominantly rural areas, small towns and so on. What we do not know, of course, is what effect the spraying had in reducing morbidity, and infection in the sprayed areas. I do not see any way of readily assessing that at this point. I think it would be interesting to go back and look at the human and equine data in terms of case rates in sprayed versus non-sprayed areas. I do not know if that is possible to do. Clearly, in the future, you will need to answer the question about efficacy of spraying by means of some measurement of virus transmission before and after the spraying. Possibly, the sentinel flock baited traps would be the best way to approach that.

Comments on Manitoba's Emergency Mosquito Vector Control Program

C.H. Schaefer, B.Sc., Ph.D. Entomology

Mosquito Control Research Laboratory
University of California
5544 Air Terminal Drive
Fresno, California 93727 U.S.A.

Manitoba's emergency mosquito vector control program was very impressive. One aspect is especially noteworthy: i.e., the interdisciplinary approach to making recommendations that was followed by the Manitoba Arbovirus Surveillance Committee. Despite some reported differences of opinion, professional expertise from several areas was the basis of your recommendations to the Minister of Health. My following comments deal with the operational decisions you made after it was decided to spray those communities which were at risk of Western equine encephalitis.

First, I will comment on your choice of insecticide. As I understand it, there are 3 insecticides registered by Agriculture Canada that can be applied aerially, using ultralow volume (ULV) spray equipment, against adult mosquitoes: i.e., malathion, naled and propoxur. I believe you were correct in ruling out the use of malathion based on expected temperatures during the spray operations, assuming a decrease in effectiveness below the optimum temperature for application. However, malathion has been used extensively in warmer climates during numerous epidemics.

Naled is another organophosphate insecticide that has been used successfully to deal with mosquito-borne disease. However, this particular insecticide, as I am sure your spray operations personnel were aware, is extremely caustic and can damage spray equipment. If it is to be used, it requires that the entire spray system be stainless steel and teflon. Other materials would break down using this insecticide.

Propoxur, or Baygon, is 2-(1-methylethoxy) phenol methyl carbamate. There are dozens of insecticides like propoxur in use today. But it is not a new material. It has been in use for about 20 years. I began evaluating its efficacy in operational adult mosquito control in 1967 (Schaefer, 1968). Subsequently, propoxur was used as a replacement for some of the organophosphate insecticides to which adult California mosquitoes had developed resistance. Propoxur is also widely used throughout the world for the control of other insect pests, notably cockroaches, house flies, black flies, and many crop pests. Anecdotally, I can remember visiting oil camps in the headwaters of the Amazon in 1975 and seeing large stocks of Baygon. I understand the same is true in Africa.

Although Baygon can be used as a mosquito larvicide, its use is discouraged by most researchers because of its cost, the high application rate required and the need to keep this insecticide in reserve for those times when mosquito adults develop resistance to organophosphates.

I noted that the application rate for Baygon that was used during the 1981 emergency spray program was 0.052 kg/ha. In the U.S.A., a higher application rate is usually used: i.e., 0.08 kg/ha. We also use Baygon more often than you do in Manitoba. Instead of just one aerial treatment during a health emergency, we may use it up to 15 times per year in the same location for routine adult mosquito control.

Such applications are not done without due regard to health and safety. Besides examining the effectiveness of Baygon, we have looked at its effects on nontarget organisms and its persistence in soil, water and vegetation. We also had the opportunity of studying the effects of a major Baygon spill, as a result of an accident involving an agricultural spray plane which was carrying this insecticide. The aircraft crashed on a pasture in California, accidentally applying Baygon at a rate of 938 kg/ha. Vegetation and soil analysis were carried out over the following months. After 6 weeks, residues declined to the barely detectable level.

At an application rate of 0.052 kg/ha, the maximum deposit of Baygon, if everything aerially applied reached the ground within the spray block, would be 5.25 mg/m^2. Compared to the application rates used for residual sprays in malaria control programs (e.g., Turkey), the application rate in Manitoba is very low.

In my opinion, you had no choice but to use Baygon in your emergency vector control program. If we had to make the same decision in California, Baygon would be the insecticide that we would choose.

Second, I will comment on the way in which Baygon was applied. The Baygon MOS (miscible oil solution) formulation that you chose was developed specifically for ULV applications. It is superior to water-based formulations because of the lower evaporation rate it provides, which is especially important considering the altitude from which the product was applied.

The Beecomist nozzles used in Conair's spray system represent the latest advances in insecticide spray technology. From personal evaluation of these nozzles, I can advise you that they produce a narrow size range of droplets. Beeco makes several nozzle sleeve sizes to suit the particular type of application (e.g., 20, 40, 80 micron pore sizes). Your choice of a 40 micron sleeve was appropriate both to the product and to the height of application.

Third, the type of aircraft chosen was the right one. A large, transport-type aircraft enables the treatment of a large area during each flight. It is essential that the insecticide be applied as quickly as possible to as wide an area as possible. This would not have been possible using many small aircraft. It is not possible, even in a City that has the proper truck-mounted ULV spray equipment, to obtain this kind of coverage using ground equipment. ULV applications are totally dependent on winds to disperse the insecticide. I have seen ULV ground units go out in the Sacramento Valley, attempting to fog with malathion, Dursban and Baygon, only to have to shut down because the winds were too low to disperse the fog more than 10 m. Without the necessary drift movement, there was nothing they could do. During an emergency, it is essential that the areas at risk be treated as efficiently and as quickly as possible.

I was also impressed with the sophisticated accessory equipment used on Conair's DC-6B, including the flow control system which automatically adjusted the flow rate of the insecticide to changes in the ground speed of the aircraft. I wish we had that type of equipment in our spray aircraft because, as you can appreciate, the ground speed will change considerably as the insecticide payload is used up. The inertial guidance system, used for navigation by passenger aircraft, was also very sophisticated, enabling precision flying to and over the spray blocks.

Overall, based on the insecticide and spray equipment used and the procedures followed, I think that the emergency spray operations that were carried out were excellent.

Reference Cited

Schaefer, C.H. 1968. The University of California mosquito research program at Fresno. Proc. Calif. Mosq. Control Assoc. 36:63-65.

Comments on Manitoba's Surveillance Program for Western Equine Encephalitis

L. Spence M.B.Ch.B., MRCP, FRCP (C)

University of Toronto
100 College Avenue
Toronto, Ontario

Forecasting outbreaks of arbovirus disease can be a difficult exercise. For many years I participated in investigations on arboviruses on the Island of Trinidad, West Indies. Although many arboviruses had been found on the island along with an abundance of bird and animal life epidemics of arbovirus disease seldom occurred. All of the ingredients necessary for the occurrence of an outbreak may be present and the outbreak may still not occur. St. Louis Encephalitis (SLE) virus has caused many epidemics in the United States and also caused outbreaks in Canada in 1975 and 1976. However, this virus has never caused an outbreak of human disease in Tropical America even though its presence in that area has been recognized since 1955. The unpredictable behaviour of arboviruses presents a problem to health authorities who have the responsibility for controlling outbreaks caused by these viruses and often places them in a "no-win" situation.

In 1973, The National Arbovirus Reference Service, a Toronto laboratory associated with the Laboratory Centre for Disease Control in Ottawa, was established under my direction. Dr. H. Artsob, a federal government scientist, was assigned to this laboratory and has been responsible for many of the virological studies undertaken. Over the years the laboratory has provided reference services for the surveillance program in Manitoba. This has given us an opportunity to observe the high quality of the work done on arboviruses in Manitoba.

It is my view that a very good surveillance program has been established in Manitoba. It is important to proceed along many fronts in surveillance because one does not know in any given year which front will give the first warning of an impending outbreak. I believe that it would be useful to add wild bird serology to the surveillance program. Studies of Western Equine Encephalitis (WEE) in wild birds have been carried out in Saskatchewan.

I support the use of mice for isolation of WEE virus from mosquito suspensions. The procedure is expensive and it is sometimes necessary to pool five mosquito suspensions for mouse inoculation in order to reduce costs. I recommend that pooling should be avoided until WEE isolations have been made. Other less expensive systems such as duck embryo tissue culture should be tested for sensitivity to WEE. Original infected mosquito suspensions should be titrated in different systems. It is desirable that any system used should be capable of detecting a broad spectrum of arboviruses.

Rapid identification of virus isolates is important. The use of a 10% suspension of infected suckling mouse brain in saline as antigen in complement fixation tests will allow identification in 24 hours. Infected tissue culture fluids can also be used as antigens.

An aggressive approach should be adopted to the diagnosis of human cases of encephalitis. Blood specimens should be collected daily and tested by complement fixation with Herpes simplex and arbovirus antigens. Delay in the collection of convalescent specimens should be avoided. The same philosophy should be applied to equine cases with the omission of Herpes simplex antigen. It is difficult to isolate virus from horse brain if the horse's illness lasts more than 70 hours. It might be useful to look for virus and antibody in cerebrospinal fluid from human cases. A search for mild cases of WEE infection should also be carried out during epidemics. Special groups such as farmers could be studied for evidence of asymptomatic infection.

In spite of the availability of a WEE equine vaccine, many horses died during the 1981 outbreak. Nevertheless, horses played an important role as sentinels for virus activity. A more aggressive vaccination program could reduce equine losses in future outbreaks but this may deprive the surveillance program of an important component.

It is important to try to identify the population at risk from WEE. This is not the time for use of a human vaccine but possible vaccine use should be kept under constant review. A human vaccine might be needed if epidemics became more severe or if a group that needed to be protected could be clearly identified.

Planning for the next epidemic should be started as soon as possible because it is difficult to plan in an epidemic situation. Plans should be made to assess the effectiveness of control measures and special funding should be obtained for this. Studies of this type could result in financial savings in futures years as only control measures of proven effectiveness would be implemented.

Public education is extremely important. A film should be prepared for public viewing which should emphasize the difficulties involved in controlling WEE. A better understanding on the part of the public will lead to better acceptance of any inconveniences caused by the control measures.

Research should be carried out on the overwintering mechanisms of WEE in Manitoba and on the possible role of spring Aedes spp and other mosquito species in virus survival and transmission.

Finally, I am convinced that Manitoba has a good program of surveillance for WEE. I strongly recommend that this program be continued because I believe that WEE presents a threat to the health of humans and equines in the province.

To the editor: As people in the business of arbovirus monitoring are well aware, the prediction of impending arbovirus outbreaks is a difficult and generally thankless job. Numerous parameters must be taken into consideration including the density of vector populations (particularly C. tarsalis in the case of WEE), meteorological information, the degree of sentinel flock conversions and the occurrence of horse, or possibly even human, cases. All parameters must be studied carefully before decisions for control measures can be taken since certain parameters taken alone, e.g. high C.tarsalis numbers, do not automatically signal the occurrence of disease.

The surveillence program in Manitoba has proven to be highly successful in predicting possible outbreaks of WEE in humans and horses in 1975, 1977 and 1981. The sensitivity of current lab procedures for diagnosing human and horse cases can be evidenced by the large number of cases recognized in Manitoba in 1981 compared to other parts of North America where WEE activity was observed.

Despite the successful programs currently in place, it is desirable to strive for systems that are faster and more sensitive. Classical methods for arbovirus isolation from mosquitoes by inoculation of suckling mice are reliable but time consuming. Newer methods to directly demonstrate virus in mosquito pools show promise for the future[1] and should be pursued. In addition, various criteria for diagnosing human cases should be considered, e.g. IgM determinations, and the possible occurrence of other arbovirus infections such as St. Louis encephalitis and California encephalitis should not be excluded.

Studies should be encouraged on the determination of baseline antibody levels in human and resident bird populations of Manitoba, on possible overwintering mechanisms of WEE virus relating specifically to Manitoba, and on WEE strains isolated over a period of years in the Prairie provinces both during outbreak and non-outbreak years. Virus isolates should be examined for possible antigenic variation as well as for differences in virulence from one year to another.

Such studies could be pursued at an academic level, but data obtained may prove invaluable to surveillence programs and may provide added input when deciding whether control measures should be instituted in any given year.

H. Artsob, Ph.D.,
National Arbovirus Reference Service,
University of Toronto.

Reference

1. Hildreth, S.W. et al 1982. Detection of LaCrosse arbovirus antigen in mosquito pools: application of chromogenic and fluorogenic immunoassay systems. J. Clin. Microbiol. 15: 879-884.

Agriculture
Canada

Food Production and Inspection Branch
Direction générale, Production et inspection des aliments

Ottawa, Ontario, K1A 0C6

Your file Votre référence

September 29, 1982

Our file Notre référence
834.71M5

CRITIQUE OF MANITOBA WEE CONTROL PROGRAM

First, I should like to indicate that in my opinion the Winnipeg authorities should be commended for a well-run program and for their desire to improve their capabilities in meeting a defined medical hazard through workshops and collaborations such as this. My comments will be restricted to the areas of control and surveillance and are offered as constructive suggestions to strengthen existing programs and to indicate areas requiring further research, and not as criticisms.

1. There is a need for more rapid identification of the criteria used to identify an incipient medical emergency. This could be partially achieved by increasing (doubling) the number of monitoring stations for C. tarsalis females and the number of sites for sentinnel flocks throughout the province. Weekly as opposed to biweekly sampling would allow more rapid identification of virus and AB titers. This assumes a rapid identification by the seriology laboratory, the feasibility of which is more suitably addressed by others, and a commitment to provide increased manpower in the monitoring area.

2. A blood survey of horses within the province could determine more accurately the frequency of WEE, the epizootic loci, host age or breed susceptibility and the incidence of death from confirmed cases.

3. Stepped-up province-wide education programs outlining methods of reducing both mosquito populations and human exposure in rural areas could be coordinated with an improved transfer of information to the public; ie. release of weekly updates on mosquito numbers, AB's in sentinnels, publishing of efficacy and environmental data gathered from spray programs in local newspapers, etc.

4. There are still many questions relating to the transmission cycle which require answers. The vertebrate reservoir, avian, mammalian or both, needs investigation. Mosquito-viral interactions need study, - does the virus overwinter in the mosquito vector, how and where does it replicate, can the virus be transmitted transovarially? Research into improved control measures, biological and narrow spectrum chemical controls, should continue to be a priority. Such answers cannot come from short-term funding but require a long-term financial commitment, such as a 5 year research plan.

My final recommendation would be the formation of short and long-term goals by an appointed committee, representing major problem areas (control, surveillance, serology, veterinary and public health, etc.) and an active lobbying (municipal, provincial, federal, research foundations, etc.) for funding to meet identified objectives.

J.E. Hollebone, Ph.D.
Pesticides Division

Canada

[illegible]

OTTAWA, Ontario, K1A 0K9

[illegible]

COMMENTS ON VIROLOGICAL TESTING.

Donald M. McLean, M.D. Medical Microbiology, University of British Columbia, Vancouver, B.C. V6T 1W5.

Inoculation of suckling mice with suspensions of triturated mosquitoes or other field materials was the optimum method for isolatio of arboviruses, but tissue cultures provided a useful adjunct for virus isolation and identification. The turnaround time of about 7 to 10 days from receipt of mosquitoes to presumptive evidence of WEE virus isolation was as rapid as could be expected in any epidemic. Rapidity of obtaining virological evidence of infected mosquitoes during 1981 provided a major contribution to intelligent, effective and timely implementation of appropriate mosquito control programs which limited the epidemic spread of WEE virus in urban areas.

Current serological techniques were effective in detecting early evidence of WEE antibody conversion among sentinel chickens throughout prairie areas of Manitoba, thus contributing to effective surveillance of WEE epidemic activity.

Virus laboratory results which exerted major impact on public health measures to contain the WEE epidemic were facilitated by excellent coordination of field collection teams with laboratory personnel in entomology and virology. Collaboration between members of various teams was promoted by prompt and frequent exchange of scientific information plus results. Adequate funding plus logistical support should be ensured during 1982 and subsequent summers in order to maintain high operational effectiveness of this surveillance system.

WESTERN EQUINE ENCEPHALITIS: ONE VIEWPOINT
Editorial

Allan R. Ronald, M.D., F.R.C.P., Professor and Head, Department of Medical Microbiology, The University of Manitoba, Room 545, Basic Medical Sciences Bldg., 730 William Ave., Winnipeg, Manitoba, R3E OW3

The spectre of sleeping sickness is always present in Manitoba during our short, beautiful mosquito infested summers. During the last three epidemic years of 1975, 1977, 1981, 45 patients have had an illness which has been diagnosed as Western Equine Encephalitis (WEE). Hundreds of other Manitobans have acquired the virus but due to the absence or mildness of clinical symptoms, no investigation has been carried out and no diagnosis has been made. At least 2 patients have died as a result of encephalitis and 6 or 7 have had permanent neurological residual that prevent a return to normal living. Fortunately very few infants have been ill with WEE.

Residency in rural Manitoba and an occupation that involves outside activities, are major risk factors for acquiring the illness. Seroepidemiologic studies have not been carried out to define these risk factors more precisely. However, the risk of an individual becoming ill with WEE has been small, even for individuals at maximum risk. For instance, even the risk of dying in an auto accident is greater than the risk of dying of encephalitis at the height of the epidemic for individuals living in areas of the province where no mosquito control is undertaken. Also, an individual who smokes cigarettes heavily, lives in rural Manitoba and is exposed each summer to thousands of mosquitos, has a probability of serious illness from smoking is a thousand fold greater than the risk of encephalitis during a life time of exposure. We need to be concerned that so much public attention is directed towards WEE, a relatively insignificant disease compared to the individual and societal costs of motor vehicle accidents or smoking.

The major strategy for the control of an epidemic of WEE, at least as viewed by the public, has been widespread aerial spraying to kill adult mosquitoes. This costly program has been used during each of the 3 years in which WEE outbreaks occurred. Despite its widespread use in Manitoba and in other jurisdictions, no prospective study has shown that aerial spraying significantly reduces this human disease. Retrospective epidemiologic investigation in Manitoba has not shown that fewer cases occurred, subsequent to spraying, in sprayed areas compared to unsprayed areas. Perhaps, this is due to the mobility of thepopulation in Manitoba during the summertime, with mosquito exposure during leisure time spent away from urban areas. Perhaps, it relates to the paucity of cases occurring in urban areas both before and after implementation of aerial spraying programs.

However a strategy that protects individuals at least risk of the disease, is vulnerable to criticism. Unfortunately, aerial spraying programs also are divisive to Manitoban society. Arrogance and an inability to compromise has arisen on both sides of the controversy with little dialogue between the antagonists. Any program that is designed to prevent disease and that is applied indiscriminately to large numbers of individuals without their consent, requires definite proof of efficacy. Because aerial spraying has not been shown to prevent human illness due to WEE virus, politicians and scientists must be cautious in prescribing this program.

Unfortunately, no satisfactory alternative answers for the control of either mosquitoes or WEE virus are apparent. Human vaccination is not an acceptable alternative. The following suggestions, none original, may be appropriate guidelines for consideration by government and the public: i.e.,

1. Ongoing research in Manitoba to increase our knowledge of the virus, its mosquito vectors, and reservoirs, and the pathogenesis and the prevention and treatment of the disease must be adequately funded.
2. The public should be informed that risk of WEE is small compared to the many risks taken each day. This risk can be reduced further by precautions to insure personal protection against mosquitoes, particularly for infants and individuals with increased occupational exposure.
3. Mosquito control programs should be set up throughout the province using the technology and expertise present in the City of Winnipeg.
4. Environmentalists and the public at large should be represented in decision-making processes of the Manitoba Arbovirus Surveillance Committee. These individuals must be responsible for their recommendations. If decisions are controversial, majority and minority opinions must be effectively articulated. If, for instance, a minority decision is not to engage in aerial spraying and is accepted by the politicians of the day, the individuals making these recommendations must be accountable if a widespread and devastating epidemic of WEE occurs.
5. The Manitoba Arbovirus Surveillance Committee should establish criteria for accelerated mosquito control programs, particularly as it relates to effective personal protection for individuals at risk. Broad guidelines should be developed for decisions that lead to aerial spraying so that a crisis atmosphere can be avoided.

6. **The effectiveness of aerial spraying programs during an encephalitis epidemic needs to be ascertained in some jurisdiction. This will require expensive, well designed, population-based studies. However, in the absence of sound evidence, the continuing reliance on an unproven and costly program for WEE control is not an acceptable alternative.**

UNIVERSITY OF GUELPH
ONTARIO AGRICULTURAL COLLEGE
Department of Environmental Biology

GUELPH, ONTARIO, CANADA · N1G 2W1
Telephone (519) 824-4120

Rerum
Cognoscere
Causas

July 6, 1982

TO: Members, Manitoba Arbovirus Surveillance Committee

In many regards, 1981 was not a good year relevant to Western Equine Encephalitis and its impact on the Province of Manitoba. The number of human and horse cases, the vast acreages sprayed during the emergency and the financial burden this entailed to the province bear testimonial to this.

However, 1981 was an excellent year for the Province of Manitoba relevant to how the members of the Arbovirus Surveillance Committee responded to the impending outbreak of Western Equine Encephalitis. Those members, with diverse backgrounds, differing personalities, representing different agencies moulded together to form a strong, united and productive team. This team should be congratulated for their perseverance, integrity and willingness to consider all views relevant to the 1981 situation as it developed.

I was priviledged to attend one of your committee meetings and work with a number of members during the emergency program. The experience which I received was invaluable. I'm sure each of your committee members would express similar sentiments. In this regard, the Province of Manitoba gained some tremendous assets which will hopefully be of future benefit.

The proposed text, "Western Equine Encephalitis in Manitoba" will reflect the calibre and integrity of your members. It will be of great value to all personnel involved in arbovirus monitoring and prevention. My compliments, for completing a difficult task so ably.

Sincerely,

G.A. SURGEONER
Associate Professor.

GAS/ss

Alberta
ENVIRONMENT
Environmental Protection Services
Pollution Control Division
Pesticide Chemicals Branch
403/427-5856
Telex 037-2006 TWX 610-831-2636
Oxbridge Place
9820 - 106 Street
Edmonton, Alberta, Canada
T5K 2J6

September 17, 1982

Dr. L. H. Sekla
Manitoba Health Services Commission
Cadham Provincial Laboratory
750 William Avenue
P.O. Box 8450
Winnipeg, Manitoba
R3C 3Y1

Dear Dr. Sekla:

OPINION OF MANITOBA'S WEE SURVEILLANCE AND CONTROL

I will restrict my comments to those areas of vector control and surveillance only, since my expertise is very limited in areas specific to epidemiology and virology.

As an overview of the Manitoba WEE Surveillance and Control Program (including the workshop), I must provide a testimonial as to the comprehensive and efficient manner in which it is conducted. However, I do have a few specific suggestions/comments for consideration by the WEE Program Co-ordinators.

First of all, I fully endorse the conclusions and recommendations as put forward by the Vector Workgroup (February 19, 1982); I emphasize the importance of rapid viral isolation/diagnosis of mosquito pools. To this end, it is imperative that emphasis be placed on processing of *Culex tarsalis* pools prior to processing pools of *Aedes sp*. adults.

Secondly, I suggest that surveillance of *Cx. tarsalis* larval populations in rural communities be included within the context of ongoing *Cx. tarsalis* population studies. Information generated from such surveillance can only enhance the predictability and extent of adult population 'outbreaks' throughout the province.

With regard to the control aspect of the Manitoba WEE Program, I stongly urge that greater emphasis be given to a ground adulticiding program (particularly for rural communities) as an alternative to the aerial adulticiding program presently employed. I have yet to be

. . . . 2

convinced that aerial adulticiding provides greater efficiency in controlling adult mosquito populations in areas that are accessible by ground adulticiding vehicles. In addition, ground adulticiding methods i) provide a greater flexability in that "environmentally sensitive" areas can be eliminated from the spray route; ii) are less dependent upon 'ideal' meteorological conditions during spray program; iii) allow for increased accuracy in targetting insecticide spray to adult mosquito resting sites; and iv) are less 'cost prohibitive' than are aerial spraying methods. Because of the above factors, it is feasible for smaller communities to own their own equipment and to deal effectively with adult mosquito populations in and around the community.

A second suggestion, related to vector control, is that adulticiding efficacy (by means of 'bite' counts and light traps as practiced by the City of Edmonton, CO_2-baited traps, or caged-mosquito studies) should be assessed regardless of whatever adulticiding technique is used. The benefits of efficacy measurements are too numerous to include herein.

One final comment that I do wish to include within the text of this letter relates to the possible creation of a Biting Fly Control Co-ordinator for the Province of Manitoba. The appointment of an individual to such a position could only serve to enhance the transfer of technical and practical information to communities outside of Winnipeg and Brandon (where expertise is presently isolated). By working with and through members of the rural communities, the incumbent of the position could provide the vehicle for dissemination of information and technical skills which could go a long way in providing self-sufficient mosquito control for each community. The question of gainful employment to which the co-ordinator could address him/herself during the winter months may not be the problem that Dr. Neufeld noted during his summary remarks to the WEE Workshop. I can soon attest to the inordinate amount of time required to organize training courses for pesticide applicators, to visit and acquaint town/city councils with details of mosquito control programs and costs arising thereof, to answer public inquiries/complaints specific to mosquito control measures, and the list goes on and on! I would further suggest that the creation of such a position would, in part, relieve the burden of responsibility for such matters which are presently delegated to researchers Drs. Brust, Ellis, and Chance. The time and effort of these individuals would be more profitable to all concerned if spent in areas of research and development of new scientific information.

Thank you for the opportunity to express my views in this matter.

Yours truly,

B. W. Taylor
Co-ordinator
Alberta Biting Fly Control Program

BWT/bld

Alberta

SOCIAL SERVICES
AND COMMUNITY HEALTH

In Replying Please Quote:
B 062.1
Seventh Street Plaza
10030 - 107 Street
Edmonton, Alberta, Canada
T5J 3E4

September 9, 1982

Dr. L.H. Sekla
Assistant Director
Cadham Provincial Laboratory
P.O. Box 8450
Winnipeg, Manitoba
R3C 3Y1

Dear Dr. Sekla:

Thank you for the opportunity to provide my views on the Manitoba Arboviral Surveillance Program. As you know, I was closely associated with the program from its inception in 1975 until I left Manitoba in 1980.

Western Equine Encephalitis is a disease which occurs in Manitoba at irregular intervals, occasionally in what could be considered epidemic proportions. Its impact when it does occur can be significant.

It is, therefore, worthwhile to maintain a surveillance program to detect the presence of the disease in the province in order that some preventive action can be taken. The Manitoba program of monitoring mosquito activity, presence of virus in mosquitoes, transmission to birds (through sentinel chicken flocks) and cases in horses and humans appears to have been successful.

The program has identified when virus is present and has accurately predicted whether or not a problem was likely to occur in each of the years since 1975. Early detection of a potential problem allowed information and advice to be provided to the population so that they could take appropriate action to avoid mosquitoes.

With regard to the aerial spray campaigns, I believe they have been effective in reducing the risk of human disease but I doubt if it will ever be possible to document their effectiveness nor the degree of protection provided.

.../2

I would support the continued operation of a surveillance program.

I trust these brief comments will be of some value to you.

Yours sincerely,

John R Waters

J.R. Waters, M.D., F.R.C.P.(C)
Director, Communicable Disease
Control and Epidemiology

/ss

CHAPTER XI :

Recommendations for Future Surveillance and Control Programs on WEE in Manitoba

Vectors

5. Research is needed to determine which mosquito species most commonly infected with WEE virus in Manitoba, are best able to transmit the virus from a bird to a mammal, under laboratory conditions.

   Although Culex tarsalis is believed to be the primary vector of WEE, several species of mosquitoes are found naturally infected with WEE virus in Manitoba. The highest ratio of WEE virus isolations from mosquitoes has been as follows: Anopheles earlei, Mansonia perturbans, Culex tarsalis, Culiseta inornata, Aedes vexans. Research is needed to determine the relative importance of these species in WEE transmission.

6. Research is needed to determine if mosquito vectors are able to transmit WEE virus transovarially to the next generation, and if mosquitoes are potential overwintering hosts for the virus in Manitoba.

   In recent years, CE virus, Japanese B Encephalitis virus and SLE virus have been shown to pass transovarially to the next generation in vector species of mosquitoes. Research is needed to determine if WEE virus can be passed to future generations in the potential vector species.

Reservoirs

7. A serosurvey for antibodies to WEE virus in wild reservoirs in Manitoba is needed.

   Manitoba is an endemic area for WEE, and one or more equine cases occur nearly every year. There have been 3 human epidemics over a 6 year period (1975-81). Birds, mammals, reptiles and amphibians have been suggested as reservoir hosts for WEE virus in Manitoba, but no comprehensive serosurvey of these animals has been attempted. One hypothesis states that migratory birds bring WEE virus north each spring, from an endemic area in the U.S.A.; another states that the virus overwinters in Manitoba in some manner. The serosurvey would provide a mechanism for testing these theories.

Control

8. A contingency plan, including a Provincial funding commitment, is needed to evaluate the effectiveness of a future aerial spray program to control WEE in Manitoba, and to monitor the environmental effects of spraying.

   The past 3 spray programs (1975, 1977, 1981) to control WEE in Manitoba were carried out on very short notice. All available resources were put at the disposal of the surveillance committee to obtain information on WEE virus in mosquitoes throughout the

Recommendations for Future Surveillance and Control Programs on WEE in Manitoba

R.A. Brust, B.S.A., M.Sc., Ph.D., F.E.S.C.

Department of Entomology, University of Manitoba
Winnipeg, Manitoba R3T 2N2

Although the causal organism of Western Equine Encephalitis (WEE) and its mode of transmission have been known for 50 years, there are many unknown factors that make the appearance and disappearance of the disease in western North America a continuing threat to public health. In Manitoba, research in the following areas is recommended:

Virology

1. A rapid method of confirming WEE isolations from mosquitoes is needed.

   Early detection of WEE virus from mosquitoes is essential to assess the risk to humans and horses. If the virus could be isolated and identified 2-3 days after the mosquitoes are processed, this would greatly improve Manitoba Health's ability to deal with an impending epidemic. The ELISA technique might be adapted to WEE virus detection in mosquitoes.

2. A rapid method for the serodiagnosis of human infections is needed. Research should be carried out, for example, on the ELISA procedure for detection of specific IgM antibodies in humans.

Epidemiology

3. A serosurvey of arbovirus antibodies in Manitobans is needed.

   One of the conditions for the development of an epidemic is the presence of an adequate number of susceptible humans. In addition to determining the percent of susceptible humans, the serosurvey would be useful in assessing the ratio of asymptomatic/symptomatic WEE infections, a ratio that is often quoted to be as high as 1000:1. In addition to WEE antibodies, the serosurvey would demonstrate the frequency St. Louis Encephalitis (SLE) and California Encephalitis (CE) antibodies in Manitobans.

4. A serosurvey of WEE antibodies in horses is needed.

   The percent of the population and the age of susceptible equines in the Province of Manitoba are important factors in a) identifying which animals should be vaccinated first in an impending epizootic, and b) for predicting the extent of the outbreak.

province, to obtain blood samples from sentinel chicken flocks, to replace the serologically WEE positive birds with negative ones and obtain supplies of insecticide.

There were insufficient resources to adequately monitor the spray program, to determine 1) the effectiveness of the spray in controlling mosquitoes 2) the effectiveness of the spray in reducing virus transmission to sentinel chickens 3) the quantity, size and dispersal of the insecticide droplets that reached the ground. This would measure indirectly the success of the spray program. 4) the effect of the insecticide spraying on non-target animals, including insects (honey bees) 5) the amount of insecticide deposited in drinking water supplies 6) the parity rates of mosquito vectors after spraying as compared to pre-spray parity rates. If the vector species had been multiparous prior to spraying but are non-parous after spraying, this means the latter have not taken a blood meal and are not involved in virus transmission.

Human Health Concerns

9. An epidemiological and toxicological assessment of the effect that aerial spraying with an insecticide has on humans should be carried out.

Effects that have been reported include, amongst others, dizziness, allergic response, respiratory disorders, and asthmatic attacks.

WEE Vaccine

10. Research should be directed towards the development of a human vaccine that is safe and effective.

To date, vaccines have been produced for experimental use only, or for laboratory workers who handle live WEE virus and who are at greatest risk. The vaccines produced to date have reduced, but not eliminated the risk of obtaining WEE.

Public Education

11. Public education programs on personal protection against mosquitoes, methods of avoiding exposure to mosquito vectors during the times of greatest risk, and methods of reducing mosquito breeding on personal property should be developed. These programs should be tested and improved to the point where they will have the greatest possible impact on WEE prevention. Such a program would greatly reduce the need for insecticide spraying, and under a lesser risk situation, provide the protection needed to prevent human cases.

Arbovirus Surveillance Program

12. A comprehensive arbovirus surveillance program must be conducted each year in the Province of Manitoba, to provide the baseline data necessary to recognize incipient outbreaks of WEE.

To maximize the effect of the WEE prevention and control program in the Province of Manitoba, a minimum of 15 locations across southern Manitoba should be monitored for WEE activity. The surveillance program should not be cut for the sake of economy, since this greatly weakens the ability of Manitoba Health to identify the 'risk possibilities' of WEE. The earlier these are identified, the greater the chance of reducing the risk to humans.

Research Funding

13. If progress is to be made on the effectiveness of a prevention and control program in modifying the natural course of an epidemic, funding for research into the annual cycle of WEE must be a high priority.

POOR

QUALITY

ORIGINAL